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STEATORRHŒA.[1]

By LAURENCE HUGHES,

Honorary Physician, Royal Prince Alfred Hospital; Honorary Physician, Royal Alexandra Hospital for Children; Lecturer in Medical Diseases of Children, University of Sydney.

STEATORRHŒA may be defined as a clinical characteristic of the stool accompanying any morbid process in which the food fat is insufficiently digested or absorbed. Etymologically the word implies a flux or flow of fat, but it may be mentioned here that frequent stools are not a necessary accompaniment of conditions associated with the improper digestion or malabsorption of fat.

During physiological digestion neutral fat is hydrolysed by pancreatic lipase to form glycerin and fatty acid, the latter being partly converted into soap by means of base derived from the pancreatic juice and bile. Bile salts increase the digestive activity of lipase and also facilitate the process of saponification. The glycerin, soap and fatty acids, dissolved in bile, pass into the villi of the small intestine, where there occurs a resynthesis of neutral fat which passes to the lacteals and thence to the mesenteric lymphatics and thoracic duct. As a corollary to these physiological facts, it follows that an excess of fat in the stools may occur: in patients with pancreatic insufficiency from inflammatory or mechanical causes, or with obstructive jaundice, owing to an incomplete digestion of fats; in patients in whom there is interference with the passage of resynthesized fat from the intestinal wall to the thoracic duct; and in patients in whom there is interference with the absorption of the products of fat digestion from the intestinal canal. It is neither practicable nor desirable to attempt to cover the whole of this field in one paper. In the

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 31, 1939.

first group passing mention may be made of the typical oily stool sometimes seen in pancreatic steatorrhœa, in which over 25% of the fæcal fat is in the form of neutral unsplit fat. If present in large amounts, it produces the so-called "butter stool". Concerning the second group, reference will be made below to instances which have from time to time appeared in the literature. It is my intention, however, to confine my remarks chiefly to the third group, and particularly to the clinical entity characterized by steatorrhœa due to a persistent malabsorption of dietetic fat, for which no satisfactory ætiology has as yet been found.

Steatorrhœa from Mesenteric Lacteal Obstruction.

Ryle[1] (1924) described cases of *tabes mesenterica* in children who clinically manifested steatorrhœa and other symptoms commonly seen in cœliac disease, and in whom there was evidence of obstruction and distension of the lacteals. In the same year Davenport Jones[2] observed lacteal obstruction during an operation for appendicitis upon a patient with sprue. Fairley and Mackie[3] (1937) described steatorrhœa occurring in two adult patients with lymphadenoma, in one with lymphoma, and in one with lymphosarcoma of the mesenteric lymph glands. *Post mortem* examination of the patient with lymphoma revealed an œdematous mesentery studded with firm mesenteric glands of varying size and the lymphatics were dilated. One of the two patients with lymphadenoma had an attack of subacute intestinal obstruction, during which numerous enlarged glands were noted in the mesentery and the lymphatics of the small gut were "enlarged and stood out like a specially injected museum specimen". Biochemical changes similar to those found in sprue and cœliac disease were also noted in these patients.

Steatorrhœa from Malabsorption of Fat.

Chronic enteritis, with its intestinal hurry and disease of the intestinal wall, may cause deficient digestion and absorption of fat, with the occurrence of fatty stools. The enteritis which may accompany abdominal tuberculosis and the chronic enteritis associated with dysenteric infections are examples. Cœliac disease in the child may for this reason be mistaken for abdominal tuberculosis.

A.C., a boy, aged three years, was well until he was two years old, when he began to lose weight intermittently, and for nine months prior to admission to hospital he had diarrhœa with several loose motions a day and some enlargement of the abdomen. There was a family history of tuberculosis and the child was a contact. The condition was considered to be abdominal tuberculosis, but further investigation proved it to be cœliac disease, and the child responded very well to treatment.

Steatorrhœa may, however, result from the malabsorption of fat in patients who have no organic lesion of the gastro-intestinal tract or abdomen and in whom there is no evidence of pancreatic or hepatic disorder. Thaysen called this condition idiopathic steatorrhœa.

Idiopathic Steatorrhœa.

My experience in this subject is practically limited to cœliac disease, and it is my intention accordingly to limit my remarks chiefly to this disorder. Thaysen[4] was of opinion that cœliac disease, non-tropical sprue, and tropical sprue could be considered one disease, grouped together under the one designation, idiopathic steatorrhœa. This view has been generally accepted on the Continent and in the United States of America. Bennet, Hunter and Vaughan[5] and other English writers have opposed this view. They consider that cœliac disease and non-tropical sprue have the same ætiology and may be grouped as idiopathic steatorrhœa, but that tropical sprue is in a different category. Thaysen's arguments are admittedly convincing, and as the three conditions have at any rate many points in common, it is convenient for the present purpose to consider them as one entity.

Historical Summary.

In 1888 Samuel Gee[6] described his classical clinical picture of "The Cœliac Affection" and recognized that the disease closely resembled certain cases of tropical sprue. Fifteen years later Cheadle[7] described a similar condition as "acholia", in the belief that lack of bile was the predominant ætiological factor. Here again it is interesting to note his remark that "the term acholia which I have been in the habit of using to indicate this condition has also been adopted by Dr. Manson to indicate the same phenomenon in the case of sprue". In 1902 and subsequent years Bramwell[8] published a series of clinical studies on "pancreatic infantilism" which have since been regarded as instances of cœliac disease. A few years later Herter[9] described the disease as "intestinal infantilism", in the belief that it was due to a chronic enteritis. His metabolic studies were a notable addition to the knowledge of the disease, and he drew attention to the increase in the stools of both total fat and split fat. During more recent years there have been a number of valuable contributions on this type of steatorrhœa, and a special tribute is due to the work of the late Hess Thaysen in this regard.

Ætiology.

The cause of idiopathic steatorrhœa is at present quite unknown. As already stated, pancreatic and hepatic deficiency, chronic enteritis and also bacterial infection have been at times considered as ætiological factors, but have long since been disproved. Moncrieff and Payne[10] (1928), as a result of estimation of blood fat in normal children and in children with cœliac disease, suggested that "the primary fault in cœliac disease is not a malabsorption of fat, but a mal-utilization of fat—a disorder of fat metabolism associated with an increase of fat in the blood and in the fæces, where possibly it is being excreted". This theory was later proved to be incorrect by Fanconi[11] and

Parsons.[12] It is now generally agreed that the essential feature of idiopathic steatorrhœa is defective absorption of fat; but there is no satisfactory evidence as to why this occurs. In recent years attention has been again directed to the action of bile in the absorption of fat and its possible importance in cœliac disease and allied disorders. According to the "choleic acid principle" of Wieland and Sorge,[13] deoxycholic acid, one of the bile acids, is an important factor in the passage of insoluble substances, such as fat and fatty acids, through the intestinal wall. Gardner and Gainsborough[14] considered this to be a more satisfactory explanation of the absorption of intestinal fat than those previously given. Mention should also be made of the recent work of Verzár.[15] He has shown that phosphorylation controls the absorption in the intestinal mucosa of fatty acid and glucose and also of flavin, a substance which helps to make up the vitamin *B* complex. The phosphorylation process is assisted by the action of adrenal cortical hormone, and adrenal insufficiency may consequently be a factor in the interference with normal intestinal absorption.

In regard to tropical sprue, I saw occasional cases of this disease during the war days in New Guinea. The accompanying stomatitis and the large, pale, foaming and offensive stools left their impression as clinical features; but I have not seen a patient with sprue since then. The disease at that time was considered to be due to an infection, probably a monilia. In 1915 and subsequent years Ashford[16] recorded important work in support of his view that sprue was caused by a special variety of yeast fungus, which he called *Monilia psilosis*. This organism, however, has since been commonly found in stools not associated with sprue, and the suggestion that it is an ætiological factor in this disease has been abandoned. Nevertheless, some recent observers still hold that there is strong evidence that there is an infective factor in sprue. Manson-Bahr[17] has noted the close association between sprue and amœbic dysentery. The two diseases may occur together, or sprue at times appears to be "grafted onto the intestinal amœbiasis". Attention has already been drawn to Thaysen's opinion that tropical sprue is in the same category as other forms of idiopathic steatorrhœa.

Symptoms.

There appears to be general agreement that there is no fundamental difference between cœliac disease and non-tropical sprue, and the symptoms of cœliac disease will be considered here. I propose to give a general text-book outline of the disease and to consider in greater detail some of the more important diagnostic features. During the last five years seventeen patients with cœliac disease have been admitted to the Royal Alexandra Hospital for Children. There were nine boys and eight girls, the ages varying from ten months to five years, the average age at the onset of the illness being two and a half years. Non-tropical sprue is regarded as a disease in which symptoms analogous to cœliac disease begin in adult life. A number of undoubted cases of this kind have been recorded by several observers. In others there is a history of cœliac disease during infancy. The onset of cœliac disease, though occasionally sudden, is more commonly insidious, with anorexia and some looseness of the bowels. The stools become large, foul smelling and pale, owing to excess of split fat, and they appear to be greater in bulk than can be accounted for by the food intake. The latter characteristic is a more constant feature than the colour and odour, for in the treated case and non-diarrhœic type of the disease the stool may be coloured and not offensive, because the fat is mainly in the form of bile-stained soap and not fatty acids. The child becomes anæmic and irritable, growth is stunted, the limbs are thin and the abdomen protuberant and soft, and the gluteal region wasted. The large abdomen is generally a characteristic feature of the disease and the enlargement is due to intestinal distension and weakening of the abdominal wall. There is not infrequently a disordered calcium and phosphorus metabolism with resulting skeletal changes and tetany. These conditions are secondary to the steatorrhœa and associated hypovitaminosis *D*. There is an increased tolerance for carbohydrate, and a low blood sugar curve in the glucose tolerance test is considered a cardinal symptom of the disease. Some of the diagnostic features may now be considered in more detail.

Steatorrhœa.

An excess of total fat in the stools is always present, and it is in the form of split and neutralized fat. There does not appear to be any

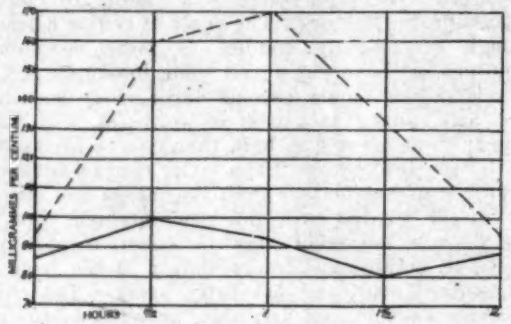
TABLE I.

Case Number.	Age of Patient in Years.	Results of Blood Examinations.			Results of Stool Examinations.		
		Erythrocytes per Cubic Millimetre.	Hæmoglobin Percentage.	Colour Index.	Total Fat Percentage.	Neutral Fat Percentage.	Split Fat Percentage.
I	4	5,990,000	50	0·4	41	6	94
II	2	4,520,000	46	0·5	54	31	69
III	3	4,630,000	56	0·6	65	9	91
IV	5	4,230,000	55	0·6	48	22	78
V	3	4,140,000	57	0·7	39	24	76
VI	1½	4,790,000	70	0·7	58	11	86
VII	2	3,430,000	52	0·7	29	19	81
VIII	1 4/12	4,780,000	70	0·7	29	11	89

inability on the part of the body to split fats, such as occurs in pancreatic steatorrhœa. According to Fowweather,[18] any stool which has more total fat than 28% of the dried matter is probably abnormal. Reference to the accompanying table will show that this condition obtains in each case, and it was present also in the other nine cases of the series. It will also be noticed that in each instance a high percentage of the total fat is split.

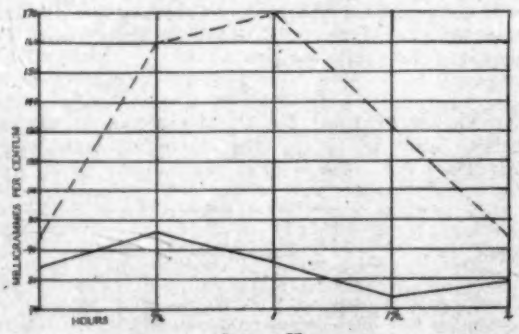
Low Blood Sugar Curve.

Thaysen[4] first drew attention to the low blood sugar curve associated with cœliac disease and other forms of idiopathic steatorrhœa. Carbohydrate tolerance tests were carried out in eight of the present series, and in each instance the curve was of the "flat" type (see accompanying graphs). The



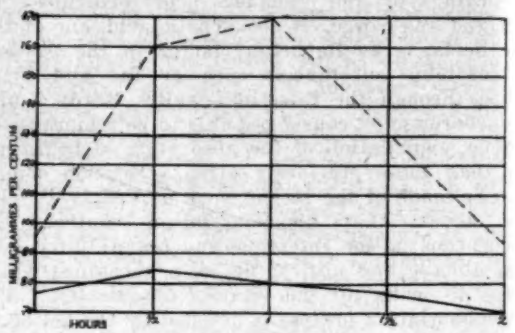
GRAPH I.

cause of this occurrence has given rise to a considerable amount of discussion. Thaysen considered that it was not associated with a defective absorption of glucose, but with an endocrine disturbance of blood sugar regulation. He supported his argument by demonstrating that the administration of an intravenous injection of glucose to these patients was also followed by a low blood sugar curve. Ross,[19] on the other hand, has recently



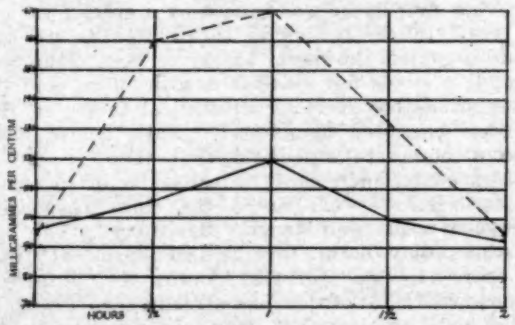
GRAPH II.

carried out sugar tolerance tests by the combined oral and intravenous methods on children with cœliac disease and found that the curves after oral administration were low and after intravenous administration high. Badenoch and Morris,[20] from studies of the absorption and utilization of glucose in children with cœliac disease, conclude that the "flat" curve occurs only in the active stage of the disease and that intestinal fermentation is an important factor in its production. In this respect it is interesting to note that the rise in blood sugar is much greater after the ingestion of banana than after the taking of an equivalent



GRAPH III.

amount of glucose. Ripe banana contains an invert sugar which undergoes fermentation much less readily than do other forms of carbohydrate. This fact has a bearing on the treatment of the disease, as we shall see later. Badenoch and Morris also incline to the opinion that the low blood sugar curve depends in part upon deficiency in pituitary contra-insular hormone, thus to some extent coming into line with Thaysen's endocrine theory. Himsworth[21] has shown that a diet which is poor



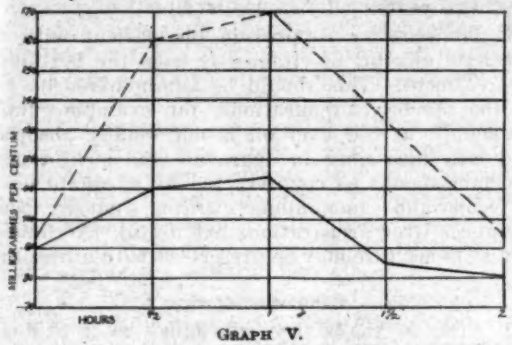
GRAPH IV.

in carbohydrate will produce a high curve, while a diet with a relatively high carbohydrate content will give rise to an increased carbohydrate tolerance and low curve. The latter condition largely obtains in cœliac disease. The balance of evidence at the present time seems to favour the view that the low blood sugar curve in cœliac disease is due to faulty intestinal absorption — that there is in fact a glycorrhœa as well as a steatorrhœa.

Anæmia.

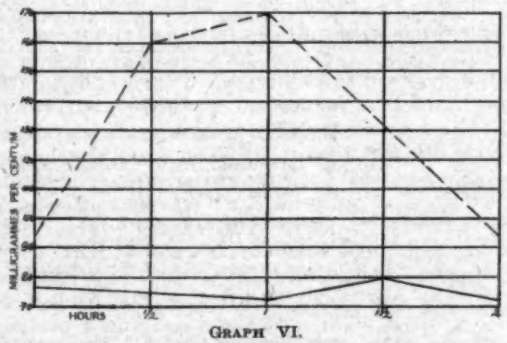
Anæmia is a frequent symptom in idiopathic steatorrhœa. Gee's original description states that: "Cachexia . . . betokened by pallor . . . is a constant symptom". Although commonly present,

the anæmia is, however, rarely the reason for which the patient is first brought for examination. In cœliac disease the anæmia is almost invariably of the hypochromic type. The red cell count may be normal and the hæmoglobin diminished, or there may be a reduction in both elements, but the colour index is always less than 1·0. These blood



GRAPH V.

findings were constant in all of the present series (see table). In tropical and non-tropical sprue the anæmia is often of the megalocytic hyperchromic type and may so closely resemble the true Addisonian anæmia as to lead to errors in diagnosis. This type of anæmia is probably due to defective absorption of hæmatopoietic substances necessary for the maturation of erythroblasts, and responds to "Marmite" or liver extract. The hypochromic type is due to defective absorption of iron and responds to treatment by adequate doses of iron.



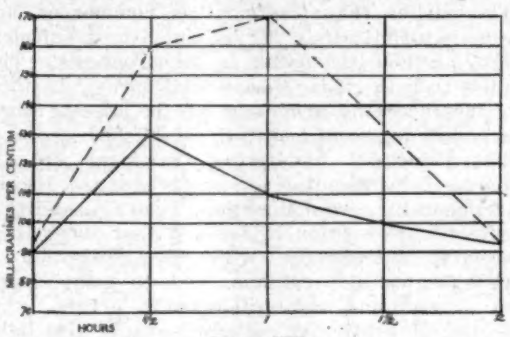
GRAPH VI.

Skeletal Changes.

Retarded growth and infantilism, which may be of marked degree, have been commonly observed since the first description of the disease. "While the disease is active, children cease to grow: even when it tends slowly to recovery, they are left frail and stunted" (Gee). During more recent years considerable attention has been paid to bony changes occurring in idiopathic steatorrhœa (Parsons,[22] Holst[23]). They consist of osteoporosis, rhachitic changes, spontaneous fractures, and bony deformities (for example, *genu valgum* and *varum*). To demonstrate the osteoporosis satisfactorily it is advisable that radiographs should be taken of the patient and of a healthy control of the same age. These skeletal changes represent clinical manifestations of abnormal calcium and phosphorus metabolism and may be regarded as a result of interference with physiological fat absorption. For a similar reason manifestations of tetany sometimes occur. It is important to stress the fact that this disturbance of calcium metabolism and the bony changes may arise in any form of steatorrhœa, whatever the cause.

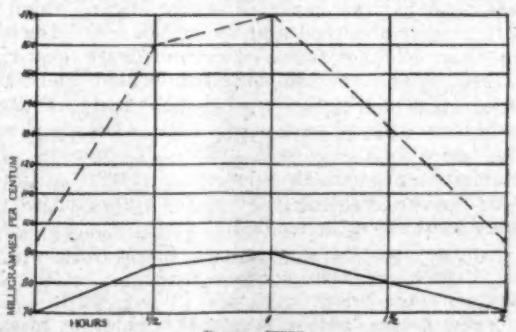
Diagnosis.

The diagnosis of steatorrhœa is suggested in any condition associated with the passage of offensive pale or greasy stools, with or without diarrhœa, the bulk of the stool being out of proportion to the food intake. It is confirmed by the finding of an



GRAPH VII.

increase of total fat in the dried stool. If this increase is due to an unduly large percentage of neutral fat, pancreatic insufficiency is suggested. If, on the other hand, it is due to an excess of split fat, the possibility of such conditions as obstructive jaundice, chronic enteritis, mesenteric lacteal obstruction and idiopathic steatorrhœa have to be considered. Overfeeding with fat, particularly in the rhachitic child, may also cause a steatorrhœa



GRAPH VIII.

of this type. In the steatorrhœa associated with pancreatic lesions there is an increase in the excretion of nitrogen in the fæces and the urinary diastase may be increased. The diagnostic importance of a low blood sugar curve in idiopathic steatorrhœa has already been stressed. In addition to these biochemical findings there are other

arresting clinical features which are of diagnostic value. In cœliac disease the stunted growth, atonic musculature, prominent abdomen, often in marked contrast to the wasted buttock, and the miserable temperament and altered disposition are common findings. In tropical sprue the stools are foaming in character and associated with diarrhœa. There is almost invariably some degree of stomatitis, and hyperchromic anæmia is common. Mild cases of this disease with stomatitis and hyperchromic anæmia may be difficult to differentiate from pernicious anæmia. The presence of free hydrochloric acid in the gastric juice, the absence of an indirect Van den Bergh reaction and leucopenia and the poor state of nutrition are important points.

Treatment.

The earlier that treatment is commenced and the more strictly it is carried out over a sufficient length of time, the better is the prognosis. This applies to both cœliac disease and sprue. In these circumstances the symptoms of the disease abate and a fair degree of health is established.

The principles underlying treatment are the outcome of the essential known facts of the disease. It is generally agreed that the primary disability is a defective absorption of fat and that there is an associated interference with the absorption of carbohydrates and calcium, and to some degree also of vitamins and anti-anæmic factors. The defective absorption of carbohydrates is also liable to cause fermentation and consequent aggravation of diarrhœa.

The first essential, therefore, is the introduction of a fat-free diet, or at least a diet from which all frank fat, for example, cream, butter, meat fat, egg yolk, is excluded. As milk forms a large part of the child's diet, it must be given in the form of protein milk or skimmed milk. It is necessary to reduce the carbohydrate intake, particularly in the initial stages. If sweetening is needed, saccharine may be used. As banana contains invert sugar, which is more readily absorbed and undergoes less fermentation than do other forms of carbohydrate, over-ripe mashed banana is often well tolerated and is a useful form in which to give carbohydrate. Protein foods form a large part of the diet. Cottage cheese (the curd of skimmed milk), flavoured with "Marmite" or tomato juice, is a useful adjunct. With these principles in view, the following serves as a useful basis for an initial diet:

Breakfast: Protein milk, cottage cheese, rusk with a little honey, coddled egg white.

Mid-morning: Protein milk.

Dinner: Cottage cheese, skim milk, egg white, mashed banana.

4 p.m.: Orange juice.

Tea: Protein milk, rusk with honey, cottage cheese, egg white, mashed banana.

When the symptoms of alimentary disturbance have abated, additional food may be added to the diet. These should at first be mainly protein, such as scraped meat, minced chicken or fish, and then additional carbohydrate in the form of vegetable *purée* and fruit and dextrinized starches, such as well-toasted thin bread and "Cornflakes". The addition of fat to the diet must be cautiously undertaken and its effect closely watched, as relapses render the prognosis less favourable. A dietetic régime of this kind should be insisted upon for at least two years.

It is necessary to provide the patient with an adequate amount of vitamin *D* from the beginning of treatment. This should be administered as one of the non-oily preparations, for example "Radiostoleum", as cod liver oil is not readily absorbed and may also tend to aggravate the steatorrhœa. Anæmia should be counteracted by adequate doses of iron and ammonium citrate, and in some instances liver preparations are useful. A rhubarb and soda mixture may be prescribed with advantage.

Acknowledgement.

I wish to thank my colleagues at the Royal Alexandra Hospital for Children for access to patients under their care, and also the staff of the pathological department at this hospital for the hæmatological and biochemical details in this paper.

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STEATORRHŒA.[1]

By F. S. HANSMAN,
Sydney.

MY contribution to the meeting will be a consideration of the physiological phenomena concerned with the ingestion, digestion, absorption and excretion of fats. No nomenclature of fatty substances has so far been unanimously adopted. The terms "lipoid" and "lipid" are two names used to include substances of a fatty nature — that is, substances which are characterized by solubility in so-called fat solvents, such as ether, alcohol, chloroform, acetone, benzene *et cetera*. Three main classes of lipoids are recognized: the true fats and oils, lipides or lipins, and unsaponifiable substances, such as the sterols and certain pigments.

The true fats have two essential components, (*a*) fatty acids, (*b*) glycerol. A fatty acid is a straight-chained hydrocarbon made up of linkages of methyl groupings with a terminal carboxyl grouping. If the carbon atoms are all satisfied in relation to hydrogen, we have a saturated fatty acid, of general formula $C_nH_{2n}O_2$. If one or more of the carbons has a double bond, that is, if there are too few hydrogens, we have an unsaturated fatty acid. According to the degree of unsaturation, the fat is in general unstable or reactive, is more oily, and has a higher capacity to combine with iodine—that is, has a higher iodine number. The fatty acids found in animals have in general an even number of carbon atoms, a characteristic important in relation to their oxidation in the body.

The other component of a fat is glycerol, which is a tertiary alcohol—that is, it has three hydroxyl groups. When the hydroxyl groups are each replaced by a fatty acid we get one molecule of fat; in other words, fats are triglycerides of fatty acids. The commonest fatty acids in animal fats are palmitic and stearic, which are saturated fats, and oleic acid, an unsaturated fat. In Nature the hydroxyls of the glycerol are generally replaced by different fatty acids; that is to say, one of the hydroxyls might be replaced by palmitic, one with stearic and one with oleic acid.

The Lipides.

The lipides are substances containing fatty acids or derivatives of fatty acids, and either nitrogen or phosphorus, or both nitrogen and phosphorus. Lipides fall into two chief groups, with subgroups; but for present purposes I shall mention only the phosphatides, which are substances containing fatty acids, nitrogen and phosphorus. Let us consider the important phosphatide lecithin. Lecithin is made up of one molecule of glycerol in which two of the hydroxyls are each combined with a molecule of fatty acid and the third hydroxyl is replaced by a phosphoric ester of the alcohol base choline (see Figure I). Phosphatides are now considered to play an essential part in the absorption of fat through the intestinal mucosa.

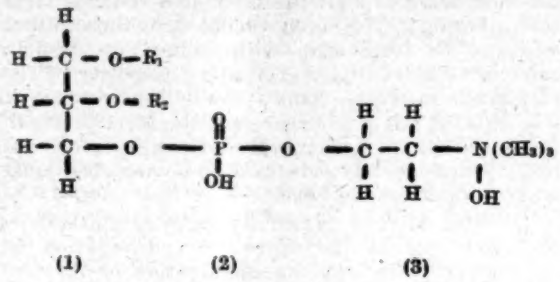

FIGURE I.
(1) glycerol, (2) phosphoric acid, (3) choline.
R_1 and R_2 are fatty acids.

The third class of lipoids are the sterols. Sterols are alcohols with the general formula $C_nH_{(2n-x)}O$, in which n is greater than 20 and x varies between 8 and 14. Sterols are widely distributed throughout the animal and vegetable kingdoms, and though they are of undoubted importance, we know relatively little of their true functions. The formula for the sterols has recently been reconstructed by Rosenheim, and the formula of cholesterol is given because of the importance of this alcohol (Figure II).

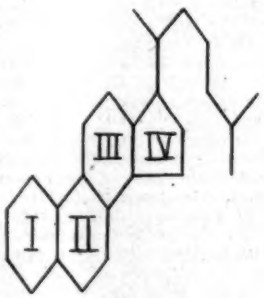

FIGURE II.

Lipoids of the Food.

Man ingests representatives of each of the groups of lipoids; they are derived from both animal and vegetable sources. For the purpose of this paper, however, I shall confine my attention predominantly to the neutral fats. In general, fats are mixed during cooking and in the mouth with carbohydrates and proteins, and this mechanical subdivision of fat particles continues in the stomach. A certain amount of fat in the form of soap is ingested. These factors, aided by lipases secreted by the stomach, tend to cause the neutral fat to emulsify. As only small quantities of chyme leave the stomach at a time, opportunity is given for the fat to be further intimately mixed with other foodstuffs, and the process of emulsification already begun is greatly accelerated by the presence of bile salts in the duodenum and the increasing formation of soaps as hydrolysis of fat proceeds. The emulsification

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 31, 1939.

of neutral fat is essential so as to provide a large surface for the lipolytic enzyme. Lipases are secreted by the pancreas and intestine, their function being to split the fatty acid radicals from the glycerol. The fatty acids are then either absorbed or form soap with sodium, potassium, calcium or magnesium. The lipolytic power of the enzymes is increased many times by the presence of bile salts. Fatty acids represent the form in which fat is soluble in water, and, as Kuhne pointed out many years ago, any substance to be absorbed must be in a water-soluble form.

Once the fat has passed through the epithelium, the next step in the process of assimilation is, according to Sinclair, the combination of the free fatty acids with phosphatides. Sinclair considers that the phosphatides present break up in such a way that the two molecules of fatty acid originally present are split off and passed into the lacteals, and their place is taken by two molecules of fatty acid that have just passed through the intestinal mucosa. In the lymphatics the fatty acids combine with glycerol to form once again neutral fats, and pass via the lacteals to the thoracic duct and hence into the general circulation. Sinclair considers that the phosphatides remain anchored in the intestinal mucosa, handing on successive molecules of fatty acids in the way described. Sinclair bases this theory on the following observations:

> By means of staining, fatty acids can be detected in the epithelial cells at the tips of alveoli within ten to twenty minutes after feeding the fat. [Laboratory animal, the rat.] By the sixth hour the epithelial cells are filled with fatty droplets with stain with Sudan III, but not with fatty acid stain, so that they appear to be entirely neutral fat. If the rat should be poisoned with iodoacetic acid or phlorhizin, the histological picture is quite different. By the sixth hour most of the epithelial cells contain considerably less fatty material than in the normal rat, and this material gives an intense reaction for free fatty acid. The same is found in adrenalectomized rats. If a cortical extract is given to the animal an hour or so before the fat, then the histological picture is quite normal. This absence of more than traces of free fatty acid in the epithelium of normal rats and its accumulation in the poisoned rat appears to fit in well with the chemical evidence that iodoacetic, phlorhizin and lack of cortical hormone interfere with fat absorption by preventing the formation of phospholipid, which has been proposed as the first step in the re-synthesis of normal fat. The results of more recent experiments involving the use of iodized fatty acids and elaidic acid leave no doubt that undigested fatty acids do become incorporated into the phospholipids of the intestinal mucosa.

This theory of Sinclair has been accepted by many biological chemists; but recently the various experimental procedures used to test the theory have been criticized by Sperry and several other investigators. They point out that iodoacetic acid and phlorhizin are general tissue poisons and do not have any specific effect on phospholipid metabolism, and that the adrenalectomized animals were moribund at the time of the experiment (this latter criticism is directed chiefly against Verzar's work). The experiments with elaidic acid are also held to be inconclusive until it is shown that on ingestion the fatty acid accumulates in the phosphatides of the intestinal mucosa before it appears elsewhere in the body.

It is certain that the absorption of fat from the intestine is not a simple, straightforward procedure; there is definite evidence that fats may be desaturated before absorption. The desaturation apparently requires the presence of certain unsaturated fats, arachnoidic acid, linoleic acid and linolenic acid, which are unable to be synthesized by several groups of mammals. Most recent work indicates that arachnoidic acid is the essential fatty acid, and that linoleic and linolenic acids are broken down to it.

I must now digress for a moment before reverting to the importance of this phenomenon of progressive desaturation of fat before absorption.

The problem of labelling fat in such a way that its future history can be studied after digestion has recently been met by the discovery of an isotope of hydrogen. This isotope is called deuterium, and consists of two atoms of hydrogen, and has therefore a mass of 2. Deuterium can replace the hydrogen in other substances; for example, deuterium oxide, or heavy water, is H^2H^2O, or D_2O. The specific gravity is 1·1056 at 25° C. Now, by the replacement of some of the hydrogen in a fat by deuterium, it has been possible to trace the fate of labelled fat in the body, and the evidence suggests that the desaturation of ingested fatty acids before absorption is considerable; there is also evidence that some oxidation takes place, with resultant shortening of the fatty acid chain. There is one group of extraordinary experiments in this regard. Shapiro, Koster, Rittenberg and Shoenheimer fed fat labelled with deuterium to a patient with a bile fistula which could be opened or closed. They report that when the bile was flowing to the exterior nearly all of the ingested fat was found in the excretions as fatty acid; but this was not the labelled fat which they had given the patient. Of the ingested labelled fatty acids about 70% were lacking, and they believe that this percentage was absorbed. Nearly the same quality of unlabelled fatty acid was excreted through the intestinal mucosa. This finding, if confirmed, presents a number of new facts, namely: (*a*) that fatty acids are absorbed almost quantitatively without bile, (*b*) that at the same time about the same quantity of fatty acid is secreted into the intestine and is reabsorbed, (*c*) that if bile is absent the fatty acids secreted into the intestine are not reabsorbed. The absorption and reexcretion of fatty acid are in this regard similar to what I think happens in the case of calcium. The evidence that I have seems to point to the conclusion that even when in the presence of calcium diarrhœa, all the calcium that finds its way into the fæces is absorbed and then reexcreted.

It has long been known that the composition of the chyle of the thoracic duct differs from that of the fat ingested, and this difference is apparently due to at least three factors: (i) desaturation of fat in the intestine, (ii) oxidation of fat in the intestine, (iii) the reabsorption of fat secreted by

the intestine. Our knowledge of the absorption of lipins, for example phosphatides and cerebrosides, is still fragmentary. As regards the sterols, including vitamin *D*, we must also await further evidence, though there is evidence that cholesterol is esterified before absorption.

Fat in the Fæces.

Having outlined the fate of ingested fat and indicated something of what is known of the part played by the intestine in the metabolism of fat, I can now turn my attention to the lipoids found in the fæces. The fæcal fat is generally considered under the following headings: total fat, soap, fatty acids and neutral fat. Total fat is the total weight of substances soluble in ether; it is generally expressed as a percentage of the dried weight of a specimen of fæces. Before it is extracted with ether, alcohol is added to render the lipids soluble. The fæces are also treated with a volatile acid, generally hydrochloric acid, so as to convert the soap present into fatty acid, for soaps are insoluble in ether. The amount of soap present is estimated by the extraction of a portion of the dried fæces with ether without this preliminary treatment with acid; the difference in the total amount of lipoids recovered gives the figure for soaps. To determine the amount of free fatty acid which is present, the ether-soluble extract is dissolved in hot benzol, and the free fatty acids are treated with alcoholic potash; from the quantity of alkali used we estimate the amount of fatty acid, each cubic centimetre of N/10 alkali being equivalent to 28·2 milligrammes of fatty acid reckoned as oleic acid. The neutral fat is then the difference between the total fat and the fatty acids.

To recapitulate: total fat is equal to the total ether-soluble material in the fæces hydrolysed by acid; soap equals the difference between total ether-soluble material after hydrolysis and total ether-soluble material without hydrolysis; the total fatty acids equal the total titrable acidity of the ether extract after hydrolysis of the fæces, and are made up of free fatty acids *plus* fatty acids present as soaps; free fatty acids are equal to the titrable acidity of the ether extract when the soaps have not been hydrolysed; neutral fat equals the total fat less the fatty acids existing either free or combined as soaps.

Having obtained this array of components we have to consider many factors before the significance of the findings of an analysis of the fat in the fæces can be understood. These can be considered under two headings: (i) What relationship does the total fat in the fæces bear to the total fat in the diet? (ii) What is the origin of fat in the fæces?

The Relationship of the Total Fat in the Fæces to the Total Fat in the Diet.—In general the fat in the diet is never considered when the biological chemist is asked to report on the fat in the fæces. In such cases the analysis is a sheer waste of time; the biological chemist performs on a small mass of casually collected fæces a long, tedious and odoriferous analysis which provides an imposing array of figures, the interpretation of which is impossible and the value to the clinician nil. Metabolic studies must be made under controlled conditions, and it is useless making extravagant calculations on what we get out of the distal end if we do not know what goes in at the proximal end. To give an opinion on the significance of a certain distribution of lipoids in the fæces, the patient should be on a controlled diet for several days before the test, and the fæces should be collected for a period of several more days on the same diet, the test period being marked off by the administration of carmine. At the same time the diet should be analysed in the same way as the fæces. Later I shall discuss what has been learnt when analyses of this nature have been carried out.

The Origin of Fat in the Fæces.—In starvation fat is always present in the fæces, to the extent of about one-third of the total dried matter. To understand the presence of fat in the fæces in starvation we must hark back to what was said concerning the secretion of fat into the intestine. This fat is apparently for the most part destined to be subjected to certain metabolic changes, probably concerned with desaturation, and then to be reabsorbed; the remainder is excreted, and, according to Bloor, part of its functions appears to be to carry off the excreted sterols. Other sources of fat in the fæces in starvation are the lipoid content of shed epithelium and the lipoid content of the bacteria. When we come to consider the relationship of the fat in fæces in a normal animal which is receiving nutrition to that in an animal which is starving, we come up against the big unknown. If I interpret the accumulated scientific data correctly, the evidence is in favour of the origin of the fats under both conditions being the same, namely, fat secreted into the lumen of the gut *plus* lipoid from epithelial cells and bacteria.

When we consider lipoid dyscrasias affecting the intestinal metabolism of fat we can do little more than use the uncertain stepping-stones of our knowledge of experimental physiology to pursue our unsteady course.

Disorders Associated with Increase of Neutral Fat in the Fæces.—In general there is such a gross excess of lipase for the amount of fat to be ordinarily digested that it must seldom happen, even if the external secretions of the pancreas are completely shut off, that fat escapes hydrolysis from lack of enzyme; thus an excess of neutral fat in the fæces is very rarely due to lack of sufficient enzyme, but usually to physical inability of the enzyme and fat to meet owing to incomplete emulsification. This incomplete emulsification may result either from deficiency of bile salts or the passage of fat in bulk into the intestine. A further factor would be interference with soap formation.

Disorders Associated with Increase of Fatty Acids in the Fæces.—A pathological increase in fatty acids, either free or as soaps, is due to a breakdown in the machinery between the lumen of the gut and

the lacteals of the villi. Whether the failure of assimilation is of the immediately ingested fatty acid or of the fatty acid arising from secretion through the intestinal mucosa, or of both, errors in phosphorylation in the epithelial cells of the mucosa are the most probable cause of an increased excretion of lipoids in steatorrhœa.

In other countries investigations have been carried out on patients suffering from steatorrhœa, in which, with a known intake of fat, the stools marked off by an indicator have been collected over a period of several days. Two diets have most commonly been used: (i) the mixed diet of Schmidt, (ii) a diet solely of milk. On the mixed diet an increased amount of fat has been generally found and the stools have had a high dry weight. On the milk diet frequently no abnormality in fat excretion is found. The Schmidt diet is quite unsuitable for Australian conditions, and the milk diet, besides being unphysiological, is unsatisfactory. There is need for a standard Australian diet for our own population, and data from normal and pathological material should improve our handling of steatorrhœa.

Conclusion.

In this paper I have refrained from a discussion of idiopathic steatorrhœa as a disease, and I have omitted any consideration of the sequelæ of the altered nutrition. The metabolism of other foodstuffs and of salts and vitamins has not been mentioned. This is undoubtedly a purely artificial approach to the subject and is justified only on the score of simplicity.

Dr. Hughes's reference to Bodenoch and Morris's theory that intestinal fermentation is an important factor in the production of the flat curve associated with idiopathic steatorrhœa prompts me to say that, as sugar is absorbed from the stomach and the first few inches of the gut, and that as the extent of the rise in the blood sugar level is not a quantitative measure of the amount being absorbed, and that as fermentation of any great degree would not be taking place until a long time after absorption, this theory is untenable.

BLINDNESS IN PRIVATE PRACTICE.

By J. Ringland Anderson, M.D.,
Melbourne.

O loss of sight, of thee I most complain!
. . . why was the sight
To such a tender ball as th' eye confined?
So obvious and so easie to be quench't.

Milton—*Samson Agonistes.*

The following analysis of the records of 12,240 patients is intended to give an accurate idea of the relative frequency of the various causes of blindness. The series is small, but it is hoped that this work and that carried out by Dr. Hamilton and Dr. Counsell in Tasmania will encourage a more systematic investigation in Australia. A detailed research into every case of blindness in the country should be made, but the most that we can do at present is to set in motion schemes of notification and examination through which a register of the blind in Australia may be completed in, say, five years' time. In the meanwhile one can endeavour (*a*) to show the necessity for a greater interest in prevention of blindness; (*b*) to show which of the causes deserve the most vigorous and immediate attack; and (*c*) ultimately to provide statistics indicating the number disabled and the extent of disablement, and thus to discover, if possible, means of improving the methods of care, education and employment of the blind in Australia.

It is difficult to know whether blindness from birth is a greater personal deprivation than the sudden withdrawal of sight in middle life, but there is no doubt which constitutes the greater economic loss to the country; and it is particularly in connexion with congenital and hereditary diseases and with those which do damage in early life that accurate statistical knowledge is necessary. It should become a powerful weapon in an attempt at public reform.

In Australia there were 3,898 blind people at the census of June 30, 1933. This was probably a low estimate, as figures from other countries show that the total obtained through a census is lower than that through inscription. The cost of pensions for the blind has increased from £95,680 to £137,150 in the last ten years. This means a total of over £2,000,000 for the next ten years. Those who are blind at an early age may receive valuable instruction at a Government institution. The State Government grant to the Royal Victorian Institute for the Blind (Melbourne) for the year ending 1938 was £2,300. The revenue of the institute from other sources was approximately £25,000. Many of the blind receive free railway and tramway passes, which in Victoria alone cost the Government approximately £15,000.

According to the age of onset of blindness, each blind person represents not only an expense but the loss of a wage earner to a household. The total number of the blind represents a great burden on the community and untold hardship on individuals.

Though ready to admit the splendid work being done by blind members of the community and the value of their example of cheerfulness, yet we cannot allow this or sheer inertia to prevent the fullest realization of the facts that most blindness is avoidable and that its prevention is our duty.

Classification.

As no ideal system of classification has been produced, the one suggested by the British Prevention of Blindness Committee has been slightly modified and adopted.

The main omission is "primary cataract". It appeared to me unwise to class as "blind" those patients with a condition so amenable to treatment. Therefore I have included only cases of operative failure or of cataract accompanied by some other

ocular or some general disease that would jeopardize the results of operation. Instead of using the heading "other errors of refraction" cases of blindness due to anisometropia, hypermetropia and astigmatism have been inserted under "amblyopia" when thought fit. Separate sub-headings have been added for "sarcoma of the uveal tract", "retinoblastoma", "orbital tumours" and "deficiency diseases".

Definition of Blindness.

So many factors are concerned in the question of the ability and the willingness of partially sighted, and for that matter, of normally sighted people, to earn a living that a satisfactory definition of industrial blindness is not a simple task. It has been convenient in this work to classify as blind those with vision less than $^6/_{36}$, or visual fields reduced within 10° in both eyes, or with less than $^6/_{36}$ in one eye and a field of less than 10° in the other. Patients with pure hemianopia were excluded. As it is impossible by studying only those with both eyes affected to realize the extent of the influence of the agents of blindness, a study of patients with unilateral blindness, that is, vision of less than $^6/_{60}$, or a field of less than 10° in one eye, has been made. Though these are not pensionable nor necessarily unemployed, their efficiency is lessened, their risks in workshop and traffic are increased, and the possibility of involvement of the remaining eye is great enough to place them at least half-way towards a pension and to being a burden for household and State.

Incompleteness of the Series.

As only patients in private practice are included in this series, it will not portray a complete picture of the total blind in this community. It is inevitable that a larger proportion of accidents, of diseases associated with poverty, with diet deficiency and other forms of malnutrition and with venereal disease will be found in a series of public hospital cases. With these exceptions the present series should give a reasonably accurate idea of the relative frequency of the causes of blindness in Melbourne. It should be emphasized that many patients, particularly those with glaucoma, were on the borderline, and it was realized that their days of earning were rapidly drawing to a close. The relentlessly progressive nature of this disease in many of the patients omitted would bring them into the limits of this investigation if it were continued for a year or so.

In the classification of the cases in this series an attempt was made to avoid such categories as abiotrophies, degenerations and senility. The first is a theoretical concept that promotes inertia rather than investigation; the other two do not refer to the underlying cause. Difficulty was experienced in separating many of the "vascular" and "focal sepsis" cases. It was thought that underlying the disease of the vessels that permitted retinitis, œdema and hæmorrhage there was a nebulous though vital degree of autointoxication.

In the following analyses the numbers under the binocular and monocular headings are not exactly comparable. A total of 9,150 patients only was considered in the collection of the monocular series, whereas records of 12,240 patients were examined for binocular blindness. The percentages against the headings of each section represent the proportion of binocular blindness attributable to this cause. Where "½" appears it indicates that of a patient blind in both eyes; only one eye was affected by the disease in question.

The investigation into the causes of monocular blindness emphasizes certain very potent agents of blindness that are not given due attention if only bilateral blindness is studied. These are particularly the amblyopia of squint, retinal detachment and all forms of traumatic blindness, particularly the non-industrial type. Table I contrasts the frequency of these causes with several others.

Table I.

Causes.	Monocular Blindness.	Binocular Blindness.
Amblyopia of squint	8·7	0·2
Retinal detachment	6·4	1·6
Non-industrial trauma	14·4	2·2
Industrial trauma	4·8	2·2
Sepsis	4·8	6·0
Vascular disease	15·1	9·5
Myopia	6·2	10·7
Primary glaucoma	16·5	20·5

Analysis of Causes of Blindness.

The causes of blindness are analysed in Table II. There were 275 cases of binocular blindness (2·2% of 12,240 patients) and 504 cases of monocular blindness (5·5% of 9,156 patients).

The several groups shown in Table II will be considered *seriatim*.

Congenital and Developmental Causes.

Congenital and developmental causes are responsible for 12·2% of binocular blindness.

Congenital, Hereditary and Developmental Defects.

Table III is an analysis of those cases due to congenital, hereditary and developmental defects.

Inheritance, Consanguinity, Immigration.—Unfortunately, little information was collected concerning inheritance and consanguinity in this series. Such information should be collected and handed to some interested person or to a registrar of blindness. Bickerton (1934) found that about one-quarter of the total blind in Great Britain suffered from hereditary ocular diseases. Hamilton and Counsell (1937) estimated that over one-third of the blindness in Tasmania was inherited.

It is significant that while only in one instance was a parent of a patient with *retinitis pigmentosa* similarly afflicted, yet four of the five patients whose pedigrees are known had normal parents or grandparents who were first cousins. Four patients with chorioideræmia had similarly related ancestors. The risks in a marriage of cousins, even when both are apparently perfectly healthy, should be more

TABLE II.

Group.	Binocular Cases.		Monocular Cases.		Requirements for Prevention.
	Percentage of Total Blind.	Age at Onset of Blindness.	Percentage of Total Blind.	Age at Onset of Blindness.	
		Years.		Years.	
A. Congenital and developmental causes—					
(1) Congenital and hereditary	12·2	0	1·8	0	Eugenic research and publicity.
(2) Myopia	10·6	50	6·2	5	Public education.
(3) Other errors of refraction	—	—	—	—	
(4) Glaucoma	20·5	39	16·5	60	Public education and research.
(5) Cataract	2·4	69	1·6	66	Improved technique.
(6) Detachment of retina	1·6	68	5·4	43	Early recognition.
(7) Retinoblastoma	—	—	0·4	2	
(8) Sarcoma of uvea	0·2	64	1·2	44	
(9) *Amblyopia ex anopsia* (strabismus and anisometropia)	0·4	?	8·7	?	Public education. Early treatment.
B. Infectious and bacterial causes—					
(1) *Ophthalmia neonatorum*	1·5	—	0·2	—	
(2) Gonorrhœa	—	—	0·6	?	
(3) Syphilis	6·9	22	1·2	20	Public education.
(4) Trachoma	3·6	30	1·6	33	Public education and research.
(5) Local infection of coats of the eye	—	—	—	—	
(6) Smallpox	—	—	—	—	
(7) Meningitis	—	—	—	—	
(8) Tuberculosis	—	—	—	—	
(9) Phlyctenular keratitis	0·7	?	1·0	?	
(10) Acute septicæmia	—	—	—	—	
(11) Focal sepsis, autointoxication	6·0	53	4·8	37	Public education.
(12) Other infections	0·4	63	1·0	47	
C. Traumatic and chemical—					
(1) Industrial—					
(a) Trauma	—	—	4·8	36	Public education.
(b) Disease	—	—	—	—	
(2) Non-industrial—					
(a) Trauma	2·2	29	14·4	20	Public education.
(b) Systemic poisoning	2·2	69	0·2	68	Public education.
(c) Birth trauma	0·4	—	2·6	—	Improved technique.
(d) War trauma	0·4	21	0·5	23	
(3) Indirect: Sympathetic ophthalmia	0·2	60	—	—	
D. General diseases—					
(1) Anæmia	—	—	—	—	
(2) Vascular diseases	9·5	64	15·1	63	Research.
(3) Of central nervous system	1·5	28	0·4	34	
(4) Connected with childbirth	—	—	0·4	33	
(5) (a) Brain tumours	0·4	44	0·8	49	
(b) Orbital tumours	—	—	1·4	27	
(6) Diabetes	6·7	70	1·2	68	Research.
(7) Nephritis	0·7	31	0·2	53	
(8) Other general diseases	1·1	55	0·2	41	
(9) Senile degeneration	1·1	82	1·0	72	
(10) Deficiency diseases	1·1	17	0·6	20	Research.
E. No information obtainable	3·4	—	5·8	—	

TABLE III.

Congenital Hereditary and Developmental Defects.

Condition.	Number of Cases of Binocular Blindness.	Number of Cases of Monocular Blindness.
Manifest at birth—		
Microphthalmia	—	2
Hydrophthalmia	2	2
Congenital cataract	5	1
Dislocated lenses	2	—
Optic atrophy	2	—
Albinism	2	—
Deficient macular pigment	4	—
Undifferentiated macula	1	—
Pigmented macula	2	—
Chorioidermia	2	2
Congenital amblyopia	1	1
Manifest later—		
Hydrophthalmia (infantile glaucoma)	1	—
Optic atrophy (Leber's)	1	1
Macular degeneration	2	—
Retinitis pigmentosa	7	—
Total	34	12

widely understood. Nettleship found consanguinity in 23%, heredity without consanguinity in 23·5%, and both combined in only 3% to 4% of cases of *retinitis pigmentosa*. Fortunately in over 95% this disease is transmitted as a recessive character. Two of the patients with congenital cataract were mongols, and their mothers were 37 years of age at their birth.

Many people are in need of advice as to the danger of propagating blindness. This will become a more pressing problem now that migrants from countries that are renowned for the prevalence of high myopia are pouring into this country. A number of myopes almost blind from degenerative changes have arrived here recently. These and members of families with diseases such as myotonic dystrophy should be watched for and forbidden admission.

Sterilization is desirable if the marriage of people blind from inheritable diseases is to be permitted in this country.

Myopic Errors.

Myopic errors are responsible for 10·6% of binocular blindness. Table IV is an analysis of cases under this heading.

TABLE IV.

Observation.	Binocular.	Monocular.
Number of cases	28½	31
Average age at loss of vision	50 years	5 years
Chorioidal atrophy	100%	77%
Retinal detachment—		
(a) Unilateral	3 cases	2 cases
(b) Bilateral	4 cases	—
Amblyopia	—	? 3
Average refraction	−16 D	−10 D
Average refraction of non-myopic eye	—	+0·5 D
Cornea in unilateral cases	—	Usually normal.
Birth injury	—	Signs present in 3 cases.

Of 31 patients blind in one eye from myopia, 48% had normal vision in the other eye. This is a high percentage and it is suggestive of several points, particularly when one remembers that these cases represent a very considerable proportion of the total or unilateral myopia. Two of the patients exhibited a paralysis of the superior rectus, but the normal condition of the cornea in all except one suggested that trauma at birth was not a factor of great importance. Occasionally defective vision is found in one eye that is out of all proportion to the changes in the fundus. The fundus may be quite free from degeneration and yet the corrected vision less than $^6/_{60}$. One must keep in mind the possibility of *amblyopia ex ametropia* in these persons, some of whom might benefit from occlusion in early life. If it is true that unilateral myopia does not tend to progress as rapidly during adolescence as the bilateral form, it would be of value to know the explanation.

It is possible that two of these cases should have been included under the heading "inherited syphilis". Infiltration and old vessels suggested that at one stage interstitial keratitis had been present, and probably apart from this the myopia would have been insufficient to reduce vision to the necessary degree.

There are few conditions in which inheritance plays a more important part than it does in myopia. We do not know whether it is more common in the axial type or in that in which the myopia though less is due to a misfit between the refraction of the cornea and lens, the antero-posterior diameter of the globe and the depth of the anterior chamber. An increase of the first three or a decrease of the fourth factor tends to produce a myopic state. Another point that concerns us is that the loss of vision need not vary directly with the degree of myopia. Though the macular area shows a predisposition to injury from stretching, yet the loss is largely fortuitous, depending on the exact site of the stretching and an innate resistance to the process. It has been suggested that in genealogies showing myopia and retinal detachment the former condition is not the cause of the latter (Sorsby, 1938).

Primary Glaucoma.

Primary glaucoma was responsible for 20·5% of blindness. Table V is an analysis of these cases.

TABLE V.

Observation.	Binocular.	Monocular.
Total cases	54	84
Average age at onset of blindness	60 years	60 years
Acute cases	6=11%	19=23%
Chronic cases	47	64

An analysis of warning signs of chronic glaucoma in 101 patients showed that 20% detected "rainbows" or haloes round lights, 13% were conscious of a blur, 7% suffered pain or headache of varying severity, and 61% observed no symptoms at all.

Of 104 patients who were examined during the year before consultation, 32% were observed by an optician and 29% by an oculist or physician; 39% had not been examined at all.

These findings emphasize the necessity for examination by a thoroughly trained oculist, who will not neglect perimetry or tonometry. They also are a further reminder of the seriousness of seeing "rainbows". Glaucoma is one of the diseases about which the public should be taught. We owe them this form of protection.

A remarkable fact, and one that provides further evidence of the treachery of this disease, is that of 126 patients almost blind in one or both eyes from glaucoma, acute or chronic, 87% had almost lost their sight when first examined by me. Of the remaining 13%, vision failed during a long interval in treatment in 7%, during treatment in 4%, and after operation had been refused in 2%. These figures demonstrate two points of great importance: first, that the majority of patients blind from glaucoma come to the oculist when glaucoma is very advanced; secondly, that even when the patient is under expert care the careful supervision of physician and of oculist may prove inadequate. Much consideration of the special problems by both specialists is necessary.

In addition to these cases of glaucoma there are those so directly related to cyclitis of septic origin, vascular disease and diabetes that they are included under these headings. As a rule these are difficult to treat. Of particular interest are those patients who, at their first visit, present evidence of bilateral cyclitis and glaucoma.

Cataract.

Cataract was responsible for 2·4% of blindness. Table VI is an analysis of these cases.

The success of operation on the other eye of the monocular cases suggests that the causes of the early operative failure were either inadequate care before or after operation, or technical errors during operation. These cases can be included, therefore, amongst the "preventibles".

TABLE VI.

Observation.	Binocular.	Monocular.
Total cases	14 eyes (9 patients)	9 eyes
Average age of patients	69 years	66 years
Operative failure	8 eyes	8 eyes
Glaucoma	1	2
Prolapse	1	3
Hæmorrhage	1	1
Cyclitis	1	1
Panophthalmitis	3	—
Phthisis bulbi	—	1
Inoperable—		
Sympathetic ophthalmia	1	—
Acute glaucoma	1	—
Cyclitis	1	—
Age	1	1
Macular degeneration *et cetera*	1	—
Average duration	—	7 years
Percentage success operation on other eye	—	100

Retinal Detachment.

Retinal detachment was responsible for 1·6% of blindness. Table VII is an analysis of these cases.

TABLE VII.

Observation.	Binocular.	Monocular.
Total cases	9 eyes of (6 patients)	32 cases
Average age	68 years	43 years
Myopia—		
Information available of	7 eyes	25 eyes
Myopic	57%	44%
Total myopic patients (including those in Table IV)	17 eyes	13 eyes
Trauma—		
Information available of	3 eyes	15 eyes—53%
Percentage with trauma	0	
Total trauma (including those in Tables XVI and XVII)	2	19

Though one can anticipate 75% of cures from the treatment of retinal detachment, yet 32 or 6·4% of 504 cases of monocular blindness were due to this cause. This is partly owing to delayed recognition; 19 patients did not come for treatment till at least six months after the first visual loss. The presence of myopia in 48% and of trauma in 63% of those supplying reliable information reveals in some measure the importance of these two agents. One case has been excluded because it was due to a birth injury. The average delay before the first consultation was four years. No operation was performed in 15 cases.

Of 36 failures among patients blind in one or both eyes, eight occurred before the era of Gonin's treatment. The causes of failure in the 28 remaining cases were as follows:

1. The detachment was too old and so reattachment or the recovery of central vision was hindered .. .. 13 cases
2. The patient was too old or unwell .. .. .. 5 cases
3. The patient refused operation .. .. 3 cases
4. Technique was defective .. .. 3 cases
5. The tear was too large .. .. 2 cases
6. The detachment was obscured by a cataract .. 1 case
7. Vitreous hæmorrhage .. .. 1 case

Retinal detachment tends to be bilateral in 66% of cases. However, it has been responsible for only two, or less than 1%, of our 215 cases of blindness since Gonin's form of treatment was introduced, whereas in 1929 Evans, when classifying 700 patients certified as blind, found retinal detachment in 4%.

Tumours of the Retina.

The cases of tumour of the retina are analysed in Table VIII.

TABLE VIII.

Observation.	Bilateral.	Unilateral.
Number of cases	—	2
Average age	—	2
Later history	—	Healthy 25+3 years later

Tumours of the Uveal Tract.

The cases of tumour of the uveal tract (0·2%) are analysed in Table IX.

TABLE IX.

Observation.	Bilateral.	Unilateral.
Number of cases	½	7
Average age	64	44
Sarcoma of choroid	½	5
Sarcoma of iris	—	1
Sarcoma of angle meshwork	—	1

Amblyopia ex Anopsia.

Cases of *amblyopia ex anopsia* (0·4%) are analysed in Table X.

TABLE X.

Condition.	Binocular.	Monocular.
(a) *Strabismus*—		
Number in series	2 eyes	39
Average age at onset of strabismus	—	1·7 years
Average age when oculist was first consulted	—	6·9 years
(b) *Anisometropia*—		
Number in series	—	5
Astigmatism	—	3
Hypermetropia	—	2

These figures give another demonstration of the results of delay in the bringing of a squinting child to the oculist. In many cases the loss of vision in an otherwise perfect eye is to be blamed on the family doctor who advised the parents of a squinting child to wait, not realizing that the time for the oculist to investigate a squint is as soon after it arises as possible.

It is suggested that in five cases the unequal refractive conditions led to such disparity between the retinal images that mental suppression occurred. This is a point that the oculist should keep in mind. Occlusion and glasses that equalize the size of the images may prevent this condition.

Infections and Bacterial Causes.

Ophthalmia Neonatorum.

Ophthalmia neonatorum was responsible for 1·5% of blindness. There were five cases; four were binocular and one was monocular.

Gonorrhœa.

The cases of gonorrhœa are analysed in Table XI.

TABLE XI.

Observation.	Binocular.	Monocular.
Number of cases	—	3
Age	—	?
Conjunctivitis	—	1
Uveitis	—	2

Syphilis.

Syphilis was responsible for 6·9% of blindness. Table XII is an analysis of the cases.

TABLE XII.

Observation.	Binocular.	Monocular.	Total.
Number of cases	18	6	24
Age	22 years	20 years	—
Inherited	15	4	19
Acquired	4	1	5[1]
Keratitis	12	5	17
Uveitis	7	—	7
Optic atrophy	1	—	1

[1] Note decline of acquired syphilis from thirty years ago.

It is of interest to realize how acquired syphilis has caused less blindness in the last twenty years. In a series from a public hospital, of course, it would occupy a more prominent place. Yet in 1921 there was evidence of *ante partum* syphilis in from 7·5% to 10% of women attending the antenatal clinic of the Melbourne Women's Hospital. In a comparable but more recent series the incidence was found to be less than 1% (Fowler, 1939).

No cases of *tabes dorsalis* or general paralysis were found amongst the patients under consideration. In many of the cases of interstitial keratitis chorioiditis was found, so the division into keratitis and uveitis is not accurate. Two sisters had interstitial keratitis at birth. In both the eyes became hydrophthalmic.

Trachoma.

Trachoma was responsible for 3·6% of blindness. Table XIII is an analysis of these cases.

TABLE XIII.

Observation.	Binocular.	Monocular.
Number of cases	10	8
Average age	30	40
Onset of trachoma (average)	24 years ago	21 years ago
Origin in country districts of Victoria	70%	88%

The relative unimportance of trachoma at the present time in the State of Victoria is shown by these figures. Of 28 eyes blind from the disease, trachoma was contracted before 1920 in 18. In none of the cases did it arise during the last seven years.

The freedom of Victorian city dwellers is shown by the fact that only one patient developed the disease in Melbourne, whilst 13 developed it in the country, two in Russia and one in Palestine.

Phlyctenular Keratitis.

The cases of phlyctenular keratitis, which constituted 0·72%, are analysed in Table XIV.

TABLE XIV.

Observation.	Binocular.	Monocular.
Number of cases	2	5
Age of patients	?	7 years

Phlyctenular keratitis is apparently not an important cause of blindness among private patients in Victoria.

Focal Sepsis.

Focal sepsis was responsible for 6·0% of blindness. Table XV is an analysis of these cases.

TABLE XV.

Observation.	Binocular.	Monocular.
Number of cases	16½	25
Age	53	37
No cause known	9	10
Probable cause	6	15
Teeth	1	5
Tonsils	1	1
Nasal and sinuses	1	4
"Rheumatic"	1	3
Endocarditis	1	1
Gall-bladder	1	0
Appendix	—	1?

Of 39 cases that were probably associated with septic absorption or autointoxication, localization of the site of infection was ascertained in only 21. The tonsils were blamed in two of these cases, the sinuses in five, the teeth in six. In addition, three of the patients had rheumatoid arthritis or some other joint disease. The fearful ravages of the seekers after sources of infection of twenty years ago have led to a reaction and at present there is a tendency for even obvious oral sepsis to be neglected. Recently a "tuberculous" keratitis in a very healthy new arrival from Berlin was attributed to a prized but very old crown with a large abscess hidden round its roots.

Other Infections.

In the small series labelled "other infections" blindness in one bilateral case was due to retinitis in pneumonia, in three unilateral cases it was due to *herpes frontalis*, in one unilateral case to dendritic ulceration, in one unilateral case to facial palsy and ulcer (cold), in one unilateral case to corneal ulceration in measles.

This is a composite group of various agents that are apt to be forgotten as possible causes of blindness. Very careful handling of *herpes frontalis* is indicated, for ulceration, iritis and glaucoma may occur.

Trauma and Chemical Causes.

Industrial Trauma.

In the group of "Blindness due to industrial trauma" there were no cases of binocular blindness, but 24 in which the blindness was monocular. These cases are set out in Table XVI. The average age of the patients referred to in this table was 36 years. In regard to insurance, information obtainable concerning 16 showed that only 69% were covered.

TABLE XVI.

Actual Agent.	Number of Cases.
Burn—	
Lime	1
Metal	1
Acid	1
Stone and cement	4
Wire	2
Nails	2
Steel	5
Wood	2
Coal, pliers, skewer, saw, boxing, spray	6

The occupation of the patients was as follows: farmer, 4; orchardist, 1; dairyman, 1; grocer's storeman, 2; merchant, 1; carpenter, 1; linesman, 2; mechanical engineer, 3; iron moulder, 1; sawmiller, 1; butcher, 2; paperworker, 1; tile layer, 1; boxer, 1; miner, 1; stone worker, 1.

Non-Industrial Blindness due to Trauma and Chemical Causes.

Non-industrial blindness in this group comprises that due to (*a*) trauma, (*b*) systemic poisoning, (*c*) birth trauma, (*d*) war trauma.

Non-Industrial Injuries.—Non-industrial trauma causes 2·2% of blindness. These cases are analysed in Table XVII.

The part played by trauma as a blinding agent is further illustrated in the section dealing with detachments of the retina.

Systemic Poisoning.—Blindness caused by systemic poisoning, responsible for 2·2% of blindness, is analysed in Table XVIII.

TABLE XVIII.

Observation.	Binocular.	Monocular.
Number of cases	8	1
Tobacco	4	1
Alcohol	3	—
Average age	69	63

Birth Injury.—In blindness due to birth injury, 0·4%, there was one case in which both eyes were affected; in 13 instances the injury was confined to one eye. This group would probably be larger if it were not for the facts that external signs of injuries tend to vanish and that the great majority

TABLE XVII.

Observation.	Binocular.	Monocular.
Number of cases	10 (12 eyes)	74
Average age	29 years	20 years
Horse accident	—	2
Bicycle accident	—	1
Motor accident	—	9
Chopping wood	1 eye	8
Hammering stone	—	1
Hammering steel	—	5
Hammering coal	1 eye	—
Scissors	1 eye	2
Fork	—	3
Pocket-knife	1 eye	—
Shears	—	1
Whip	1 eye	—
School compass	—	1
Pen	—	2
Acid	1 eye	1
Inhalation	—	1
Kalsomine	—	1
Dog bite	—	1
Baby's hand	—	1
Mat	—	1
Shovel	—	1
Nail	—	1
Wire	—	1
Stick	—	1
Thorn	—	1
Thistle head	1 eye	—
Bump	1 eye	2
Injury unknown	1 case	5
Cricket	—	1
Tennis	—	4
Shooting	2 eyes	6
Fireworks	—	1
Bow and arrow	—	1
Catapult	—	1
Tip cat	—	1
Thrown stone	—	3

of patients are at least twenty years of age when examined. It must be remembered that as the birth rate falls the percentage of *primiparæ* in the total number of births rises. This is exemplified by the figures supplied by Professor Marshall Allan (Table XIX).

TABLE XIX.

Year.	Birthrate.	Percentage of *Primiparæ*.
1926	21·98	27·8
1929	20·25	28·1
1932	16·86	28·6
1935	16·55	33·6
1937	17·43	35·8

As there is more risk of injury at a first birth, the incidence of birth injuries can also be expected to rise, other things being equal. Another factor of importance is the more widespread and more frequent use of forceps. In Allan's report (1928) we read that of 633 Victorian doctors interviewed, 134 estimated the incidence of forceps application in their practices as over 50%. There is an added risk of ocular injuries when patience and dexterity are replaced by undue haste and defective technique. In this connexion it is wise to recall the findings of Richmann (1938). He wrote that in a group of 531 babies the incidence of hæmorrhages was 12·2%. Amongst first-born babies it was 17·5%, as compared with 9·2% in the subsequent babies; but first-born spontaneous deliveries showed only 13·8% of hæmorrhages against 20·6% in first-born deliveries by forceps. Various birth stresses are

considered as ætiological factors. The length of labour seems to have no bearing on the incidence of retinal hæmorrhages.

Of the 342 infantile deaths in Victoria in 1936, 81, or 24%, were due to injury at birth. All these children died under one month (Fowler, 1939). It is unfortunate that no figures are available of those who are maimed yet survive.

War Trauma.—The cases of blindness due to war trauma, 0·4%, are analysed in Table XX.

TABLE XX.

	Binocular.	Monocular.
Number of cases	1	3
Average age	21	23

Sympathetic Ophthalmia.

There was only one case of sympathetic ophthalmia.

General Diseases.

Vascular Diseases.

The cases of blindness due to vascular disease, 9·5%, are analysed in Table XXI.

TABLE XXI.

Observation.	Binocular.	Monocular.
Number of cases	25½	75
Age	64 years	63 years
Retinitis	5	23
Retinal hæmorrhage only	5	8
Vitreous hæmorrhage	3	8
Macular degeneration (cystoid *et cetera*)	4	5
Disciform degeneration at macula	4½	1
Chorioidal sclerosis	1	3
Retinal arteriosclerosis and cataract	—	5
Thrombosis	3	16
Embolism	—	5
Cerebral arteriosclerosis	—	1
Glaucoma	3½	2

The varied nature of the conditions included in this group is a proof of ignorance. Though one may be justified in considering that a vascular abnormality is the direct cause of most of these conditions, the underlying cause is probably some form of poisoning.

Diseases of the Central Nervous System.

Blindness due to diseases of the central nervous system, 15%, is analysed in Table XXII.

TABLE XXII.

Observation.	Binocular.	Monocular.
Number of cases	4	2
Average age	28 years	34 years
Disseminated sclerosis	3	—
Neurasthenia (?)	1	—
Trigeminal neuralgia, resection, corneal scar	—	2

The need for the greatest care in treating the cornea after operation on the trigeminal nerve is not realized by all concerned.

Diseases Connected with Childbirth, Brain Tumours, Orbital Tumours, Diabetes.

Blindness due to diseases connected with childbirth, brain tumours (0·4%), orbital tumours, and diabetes is analysed in Table XXIII.

TABLE XXIII.

Observation.	Binocular.	Monocular.
Connected with childbirth—		
Number of cases	—	2
Average age	—	33 years
Brain tumours—		
Number of cases	1	5
Average age	44 years	49 years
Orbital tumours—		
Number of cases	—	5
Average age	—	27 years
Diabetes—		
Number of cases	20	6
Average age	70 years	66 years
Retinitis	12	3
Hæmorrhages	5	1
Cataract	8	2
Glaucoma	6	—

It is seen that diabetes is the general disease that is third only to vascular degeneration and syphilis as a cause of blindness. Its ravages would appear still more widespread but for the concentration of so many semi-private patients in the diabetic hospital clinics. The majority of patients in this series had retinitis. The average age of this group is high—70 years. The results of cataract extraction as a rule were not satisfactory. The high incidence of an almost hopeless form of glaucoma further complicates the visual future of diabetic patients.

Nephritis, "Other General Diseases", Senile Degeneration, Deficiency Diseases.

Blindness due to nephritis (0·7%), "other general diseases" (1·1%), senile degeneration (1·1%) and deficiency diseases (1·1%) is analysed in Table XXIV.

We have classed conical cornea as a deficiency disease because it has been experimentally produced in animals by a diet free from vitamin *A*. It is not known whether such a deficiency is also the cause of the disease in man, but the fact that most patients are country girls suggests that some environmental or nutritive factor may be responsible. All our attempts to trace a familial or hereditary element have had negative results.

Blindness of Unknown Causation.

Cases in which the cause of blindness was unknown are analysed in Table XXV.

It is probable that some of the cases of keratitis of unknown origin will prove to be due to vitamin deficiency. It is of interest to notice how many ulcers improve rapidly when various preparations of vitamins *A* and *B* are administered locally and internally.

TABLE XXIV.

Observation.	Binocular.	Monocular.
Nephritis—		
Number of cases	2	1
Average age	39 years	53 years
Other general diseases—		
Number of cases	3	1
Average age	55 years	41 years
Paget's disease; hæmorrhages, exudate	2	—
Exophthalmic goitre; corneal ulcers	1	—
Myxœdema; retinal hæmorrhage, glaucoma	—	1
Senile degeneration—		
Number of cases	3	5
Average age	82 years	72 years
Senile extropion, and corneal ulcer	—	2
Exudative retinitis	—	2
Macular degeneration	2½	2
Deficiency Diseases: conical cornea		
Number of cases	3	3
Average age when vision failed	16 years	? 20 years
Residence in country	2	3
Female	3=100%	3=100%

TABLE XXV.

Observation.	Binocular.	Monocular.
Number of cases	16	16
Macular degeneration, cause unknown	4	—
Pigmented macular degeneration	2	—
Keratitis	3	2
Corneal ulcers	—	2

DISCUSSION.

As seen from the preceding tables, the chief blinding agents in order of importance appear to be glaucoma, congenital and hereditary diseases, myopia, vascular disease, syphilis, diabetes, focal sepsis and trachoma.

For monocular blindness the order is glaucoma, vascular disease, trauma, *amblyopia ex anopsia*, retinal detachment, myopia, focal sepsis, and industrial trauma. Monocular blindness concerns us in four ways. It is a bar to certain trades and professions; it may impair efficiency if the other eye is not good; it means a greater risk of complete blindness, since sight depends on one eye only; and in progressive disease it is a warning that the other eye may be lost from the same cause. This last applies particularly to glaucoma, vascular disease, focal sepsis and retinal detachment. As a result of Gonin's discoveries, detachment is now a comparatively rare cause of binocular blindness. Perhaps we may some day find an equally effective treatment for glaucoma.

The high incidence of monocular trauma and amblyopia is worthy of attention, particularly as both should be largely preventible with a little extra care, and because they, like hereditary diseases, conical cornea, syphilis and trachoma, cause loss of sight so early in life. A very large proportion of blindness is due to public ignorance, a fairly large amount to our own. In our present state of knowledge one may class as preventible all permanent blindness due to cataract, retinal detachment, amblyopia, infections of most kinds, systemic poisoning and birth trauma, and a high percentage of that from glaucoma and myopia. A certain amount of loss of sight from trauma cannot be avoided, but much could undoubtedly be prevented if the public was seriously warned about possible sources of danger. The preventible causes enumerated above are responsible for 50% of blindness, and a further 30% might be preventible if our knowledge of myopia, glaucoma, conical cornea, diabetes and congenital and hereditary diseases could be extended. It is time that the medical profession should enlighten the public on the definite dangers of consanguinity. The sudden appearance of families afflicted with *retinitis pigmentosa* and chorioideræmia when parents and grandparents are free from ocular disease but are cousins, convinces them of their lack of forethought when it is too late. It is our duty not only to make good our own deficiencies but to teach the public to help themselves, by spreading among them a knowledge of eugenics, hygiene and the necessity for early treatment.

It would be wise to have a system of compulsory notification of blindness. The registrar to whom such cases would be reported would inaugurate investigation into such problems as the inheritance of blindness, the development of myopia, the prevention of injury and the arrest of glaucoma. These appear to be the four great causes of blindness that should be attacked immediately.

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In conclusion I would express my gratitude to Misses E. Agar, D. S. Mann and O. J. McNab for the help they have given me in this investigation.

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Reviews.

EPIDEMIOLOGY AND COUNTRY PRACTITIONERS.

W. N. PICKLES has collected his papers and experiences and published them in a valuable little book called "Epidemiology in Country Practice".[1]

[1] "Epidemiology in Country Practice", by W. N. Pickles, M.D., with a preface by Major Greenwood, F.R.S., D.Sc., F.R.C.P.; 1939. Bristol: John Wright and Sons Limited. Demy 8vo, pp. 118, with illustrations. Price: 7s. 6d. net.

His thesis is that there is still a great deal of doubt in our knowledge of the incubation and infectious periods of infectious diseases, and that the isolated rural practitioner has a greater opportunity than the city practitioner for the observation of the natural history of these diseases in a pure state, a thesis which he sufficiently demonstrates. The purpose of his book is "to stimulate other country doctors to keep records of epidemic disease and to put before them the unique advantages that their position gives them". For the "country doctor is able to see an epidemic in its true perspective, as he can follow it from the very first patient to the last, see the epidemic as a whole, and trace accurately the spread of infection". And, realizing that one man in general practice has not sufficient material for proof, he points out that what is required is "the pooled information from a large number of practices similarly situated".

The method of record which he uses is a simple graphic one involving very little labour; it could be used by any practitioner at the expense of a few hours *per annum*. It was shown to him by an officer of the Board of Health when they came into "fundamental disagreement" about the incubation period of catarrhal jaundice, and Pickles found that his opinions were valueless without documentary record. For eight years he has collected such records and has proved his case. In doing so he has gleaned interesting things about other infectious diseases, and his conclusions are the substance of this book.

But it is not for these things that the book is so valuable. It is in the fact that Pickles has proved that there is still scope for original research in the isolated country practice and that there is still room for Mackenzie's ideal in the general practitioner's mind. He has provided an inspiration to those whose occasional urges toward research have been discouraged by an awareness of their lack of both technique and knowledge.

The book has a literary quality which is unexpected in a text-book and is worth reading from this point of view alone. It is confidently recommended to every country practitioner whose interests are occupied with preventive work, and health boards should note it as a valuable contribution to their efforts to organize the units which do their work and on whose efficiency and enthusiasm the work so largely depends.

SURGERY OF PAIN.

THE manifestations of the most outstanding symptom in the whole semeiology of disease, that is, pain, are so protean, and its relief so important and yet so difficult to procure, that any further light which can be thrown on its causation and treatment will always be welcomed and helpful to the medical profession. In his recent monograph[1] on this subject, Professor Leriche has discussed in a most interesting manner many new aspects of pain, mainly of the intractable variety, and its treatment by surgery. Already well known for his work on the surgery of the sympathetic nervous system, he has devoted himself to an intensive study of pain, especially that associated with trauma, as a result of his 1914-1918 war material; and this book of lectures is an outspoken account of his theories and experiences, rather than a scientific treatise. In an extremely naïve and unconventional manner, he records his own misgivings and observations of the many difficulties he has had to face, and describes the steps leading up to the methods of therapy he has adopted up to the present. It is a book which has to be read and studied minutely, for nearly every page raises some problem which cannot yet be solved in the light of our present-day knowledge, and the author puts forward some ideas of his own which have to be carefully considered to be appreciated. Throughout the lectures he is ever ready to admit his shortcomings, failures and dead-ends, but continually points out that only by close observation and by the taking of full advantage of human experiments produced by trauma can any real advance be made. Thus, having nothing new to offer, he purposely omits any reference to the pains of *tabes dorsalis*, post-herpetic neuralgia, and the glomic tubercles of Masson. Similarly, pains of thalamic origin, bone pains and cervico-brachial pains are not included. The intractability of post-herpetic pains from a therapeutic standpoint are well known, and disappointment is felt that the views of such a man as Professor Leriche on this subject have not been included. Similarly, no mention is made of any drugs or physical therapy for the relief of pain.

The book opens with a discussion on the physiological pathology of painful sensibility and stresses the importance of the personality of the individual, his vasomotor and hormonal characters, in the assessing of pain. A habitually heightened vasomotor tone, disturbances of calcium metabolism in the tissues, associated with hyperparathyreoidism, the role of the suprarenals in the maintenance of excessive vaso-constriction and thyroxine are all said to result in a lack of sensory equilibrium.

A tribute is then paid to the numerous great pioneers in the investigation of pain, among them Horsley, Weir-Mitchell, Abbe, Spiller, Frazier, Cushing, Foerster and Jonnesco. Special reference is made on several occasions to the work of François Frank and his doctrine of centripetal sympathetic fibres. Leriche deplores the fact that, up to the present, surgery for the relief of pain has been devoted almost entirely to the division of sensory pathways, ever higher and nearer to the sensorium, and that their functional activities have not been attacked via the sympathetic and endocrine systems. He then details in chronological order the procedures that he has adopted since 1910 with these objects in view, from periarterial sympathectomy, arteriectomy, ramisection (whilst Hunter and Royle were advocating it for relief of spasm), ganglionectomy, splanchnectomy, partial removal of suprarenals, and, finally, local infiltrations with "Novocain", directly to scars and for sprains, or indirectly to the sympathetic ganglia. The pain with which Leriche is concerned is a real disease, affecting the whole individual and not merely a symptom, and it is absurd to refer to it as a "fortunate warning", not to be relieved, as in *angina pectoris*. These conditions and those in which the patient desires to be freed from drug addiction call for this newer surgery of pain; and the surgeon who is going to apply himself to this work must train and discipline himself to understand pain thoroughly and "not be content to be the mere secular arm of medicine".

The next chapters of the book deal with trigeminal neuralgias and neuralgias of the face. An extensive list of conditions which simulate true *tic douloureux* are discussed in a full differential diagnosis, due emphasis being placed on certain pains arising from the larger arteries of the head and neck, the sympathetic chain and psychalgias. Leriche rightly deplores the syndrome of useless procedures, and injections, and operations to which these patients are submitted, a sort of slow-motion crescendo treatment; and he strongly advocates an immediate retroganglionic neurotomy if the diagnosis is definite. Then follows an excellent and enlightening exposition of those post-traumatic spreading neuralgias which begin in the first place after some minor injury to a finger or toe, and are still present even after fore or hind quarter amputations, unless their association with vasomotor disequilibrium is recognized early. There are no signs of inflammation to indicate that they are due to neuritis, and reamputations only cause exacerbations by allowing neurogliomata to form on the central ends of the cut nerves. These pains and the secondary pains after nerve injuries are aptly described as the "Wandering Jews of Pain". Appropriate treatment to break the vicious circle of vasomotor changes and wide neurectomies with immediate nerve grafting between the separated ends are advocated, although admittedly not always successful.

[1] "The Surgery of Pain", by R. Leriche, M.D., LL.D., F.R.C.S., translated and edited by A. Young, B.Sc., M.B., Ch.M., F.R.F.P.S.G., F.A.C.S., M.D.; 1939. London: Baillière, Tindall and Cox. Demy 8vo, pp. 508. Price: 21s. net.

Next comes a description of causalgia in which Leriche claims priority in its recognition as arising in sympathetic dysfunction; and he criticizes others in true French style for not giving him the full credit for discovery. Painful amputation stumps, pains due to vaso-constriction and ischæmia, vasomotor nerve attacks, cervical rib lesions and Raynaud's disease are all fully dealt with, and appropriate treatment is discussed for each type. Leriche disagrees with Bayliss's theory of antidromal conduction in somatic nerves, and maintains that Raynaud's disease is not due primarily to an arteritis as stated by Lewis, but to a hypertonus following endocrine disturbance; it is not, he holds, to be confused with attacks of vaso-constriction occurring in a patient with *endarteritis obliterans*. When gangrene has set in, he insists on conservative measures to avoid amputation as long as possible, and truly states that "Nature does things more economically than surgeons".

The difficult subject of juvenile arteritis or thrombo-angiitis is fully dealt with and some new ideas on reversal of the flow of blood in vessels of the collateral circulation are put forward. In these circumstances the pulsations would not be felt in the peripheral parts of the main vessels, even though they might be full of blood; and the true state of the circulation can be determined only by arteriography. Resection of the affected portion of vessel is essential, as it acts as a nerve sending down vaso-constrictor impulses to the collateral and peripheral vessels. Partial suprarenalectomy may be helpful if the disease is not too far advanced. Senile arteritis is regarded in more or less the same light.

A very valuable, if somewhat revolutionary, chapter is devoted to the pain of *angina pectoris*, and physicians in general are severely criticized for not allowing their patients with this dire malady the comparatively simple relief of their pains and anxieties by means of injection or removal of their stellate ganglia, chiefly because they do not know of the available benefits. The pathology of angina and the centripetal role of the cardiac sympathetic nerves in the production of the bizarre distribution of referred pains are fully dealt with, after a criticism of Mackenzie's theory for referred pain, in general.

The book then concludes with important chapters on the pains due to cutaneous cicatrices and their relief by repeated "Novocain" infiltrations, the problem of visceral pain, and the mistaken doctrine of referred pains, and the treatment of the pains of inoperable pelvic and abdominal neoplasms. Throughout the book particular stress is laid on the important functions of the sympathetic nerves, and on the use of "Novocain" infiltrations of sympathetic ganglia in the investigation and often relief of many painful conditions.

The book is clearly and accurately printed on non-glossy paper which makes its reading a pleasure, and it loses none of its author's atmosphere in the splendid translation at the hand of Professor Archibald Young. Whilst it is a book essentially for the person who is prepared to make a thorough study of pain as a disease and to perfect himself in the application of the special therapeutic measures available, much of benefit and interest will be found in its pages by those who have to deal with pain in its many forms, especially when it follows trauma.

TRAINING FOR CHILDBIRTH.

UNDER the title "Training for Childbirth" Miss Randell has published a useful little book outlining a course of physical training which is designed to prepare the mother for the effort of labour and to induce subsequent physical restoration.[1] The work is based on the practice of many years in the massage department of Saint Thomas's Hospital, and is compiled for the guidance of midwives, masseuses, social workers and the mothers themselves. Believing that knowledge inspires confidence and control, and thus a safe and easy delivery, while ignorance breeds fear and difficulty, the author opens with a brief and simple account of the normal progress of pregnancy and labour and of the puerperium. This is followed by a detailed description of a series of exercises planned to produce the desired results. The author indicates how simple domestic tasks may be adapted to fit in with the general scheme, and she rightly emphasizes the importance of proper relaxation. The value of these procedures is recorded in a collection of "testimonies" contributed by some of those who have put them into practice. In some matters the author oversteps the bounds of her subject, as for example in the instruction to "keep the baby small" and in offering advice on the care of the breasts. And she is perhaps a little over-optimistic in the far-reaching results which she anticipates from some of the procedures advocated. Nevertheless her book will be enjoyed by all whose work lies within its field. The illustrations are excellent.

[1] "Training for Childbirth from the Mother's Point of View", by M. Randell, S.R.N., S.C.M., T.M.M.G.; 1939. London: J. and A. Churchill Limited. Foolscap 4to, pp. 165, with 118 illustrations. Price: 7s. 6d. net.

WOMEN AND SPORT.

DR. STEPHAN WESTMANN'S "Sport, Physical Training and Womanhood" should prove helpful to those doctors interested in athletics for women.[1] His experience seems to have been largely German, so that Australian and English readers may not always accept his point of view, especially in regard to internal examinations.

He considers sport for women chiefly from its bearing on the maternal function. He is emphatic that competitive, record-breaking sport is harmful to women (*sotto voce* he admits that it may injure men) and especially to maternity. He goes so far as to say that such sport may masculinize women, producing virile types with small pelves. He has apparently attended many sportswomen in labour, and he, very uncritically, attributes all their difficulties in labour to their athletic sins. That the masculine type of woman is likely to choose sport as a major occupation does not seem to occur to him; nor does he mention that the difficulties in labour described by him are not confined to sportswomen; nor does he consider or exclude other possible causes. He forbids sport, especially competitive sport, during menstruation. In discussing these problems he describes some individual cases, but he gives no real statistical consideration to the question. His long bibliography is less useful than it might have been, owing to the lack of indication as to the authority on which his various statements are based. The book is too dogmatic, too uncritical, and provides too little evidence to satisfy the medical practitioner. This does not mean that the book will not prove valuable to medical readers, for it deals very capably with the practical side of sport. The author believes that the gradual increase to a climax followed by a gradual decrease of the labour pain is to be taken as the curve to which the muscular efforts of women should conform in their sports and exercise. He uses this standard in describing the various sports in detail. He gives a very full description of the bodily action in various games and activities, and in criticizing or approving of them for women he always bears this curve in mind.

He approves of swimming, skating, cross-country running, walking and rowing for women, and of gymnastics and eurythmics for the expectant mother; he describes very valuable exercises for the pregnant and puerperal woman. He regards sport as dangerous in the first three months of pregnancy, being very liable to lead to miscarriage. He strongly disapproves of diving from springboards, motor-cycling, cricket and jumping for women.

[1] "Sport, Physical Training and Womanhood", by S. K. Westermann, M.D., L.R.C.S., L.R.C.P., L.R.F.P.S., with a foreword by Sir Kaye Le Fleming, M.A., M.D.; 1939. London: Baillière, Tindall and Cox. Demy 8vo, pp. 237, with illustrations. Price: 12s. 6d. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 4, 1939.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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A CALL TO THE MEDICAL PROFESSION.

THE present war has so often been described as a "queer sort of war" that the phrase bids fair to become a *cliché*. The geographical situation of Australia and the uncertainty of international relationships have combined to make it appear that Australia is somewhat slow to take an active and adequate share in the struggle, though the Commonwealth Government lost no time in proclaiming a state of war with Germany. But Australia has not been idle and we may be certain that the actions of the Government have been dictated by common sense and wise judgement. A second Australian Imperial Force is being formed, and, as has been announced in several recent issues of this journal, the medical profession is being asked to provide its share of the personnel. Vacancies still remain to be filled in two base hospitals and one casualty clearing station. Most of these vacancies are in the junior positions; these carry the rank of captain and are equivalent to the positions of resident medical officers of hospitals. There are also, as was stated in last week's issue, some vacancies amongst senior positions to be filled by physicians, surgeons and specialists.

Approaching the future in a more sober spirit and with less of the *élan* of 1914, we realize that necessity demands the imposition of inevitable hardships. It is unavoidable that in times of war there should be periods of idleness for army medical officers; but we anticipate that all possible steps will be taken to minimize such periods. We are told that the Australian Imperial Force will undergo training in Australia for at least some months. Medical attention for the troops will be required from medical officers both in the field and in military hospitals. To field medical officers the work entailed may often appear trivial and wearisome; but it should be remembered that an officer in such a position has an unparalleled opportunity of living up to the best traditions of his profession. There is much more for him to do than the mere holding of sick parades and the ordering of medicine. Not only can he superintend the bodily health, the physical well-being, of a mass of Australian youth, but he has here the chance of acting as their guide and friend and of piloting them along proper channels. The men among whom his lot will be cast will be young, eager, well fed and full of energy; they will enjoy much leisure, and clearly this leisure may be productive of good or evil. Directed aright, leisure may be used to raise the morale of the men. Among the troops may be found men with gifts of all kinds—teachers, scientists, experts in skilled crafts and industries, and so on. By proper organization not only can a healthy rivalry in open-air contests be promoted and physical condition improved, but knowledge may be deepened and education encouraged. An attitude of dissatisfaction is not infrequently noticed in medical officers attending militia camps. An outlook such as we have indicated may serve to dissipate some of this feeling and provide an objective that may be worthily sought. To those engaged in military hospitals, in contrast to medical officers in the field, will be given opportunities of more technical and professional work. But, though the senior practitioner may find the prospect of a prolonged period under these conditions depressing, both he and the raw recruit have much to learn of the military machine. It may be assumed that arrangements will be made to call up for duty, when this is necessary, the smallest number of

medical practitioners meeting requirements. In regard to seniors, it may be possible to arrange for relief at intervals, and there is some likelihood that the hospitals will be situated in proximity to capital cities and that the difficulties of the attached staff may thus be lessened.

A problem of great importance to every man joining the Australian Imperial Force, be he medical practitioner or not, is his economic position. To most practitioners, senior or junior, enlistment will entail much sacrifice. It should therefore be widely known that different groups of practitioners are making plans by which some of these financial hardships may be lessened and the burden of worry reduced. Now that the medical profession in Australia is being called on once again to take its part in the defence of the Empire, there is no doubt that it will take full advantage of the opportunity. In the Great War of 1914-1918 the Australian Army Medical Corps earned a high reputation; the honourable tradition then established must at all costs be maintained.

Current Comment.

AURAL AND PARANASAL INFECTION IN SWIMMERS.

THE occurrence of infections such as *otitis media* and sinusitis in relation to swimming and diving is quite well known. The enclosed swimming bath has been in the past a well-known offender and many investigations have been carried out to prove that infection of the water soon takes place and may quite easily be passed on to swimmers in the bath. This danger can be very greatly minimized by correct treatment of the water by some method such as free circulation combined with chlorination. In some instances it has been claimed, particularly by the lay public, that sore throats have occurred not infrequently after swimming in baths where such methods were employed, but probably in these cases all that was experienced was a certain degree of transient irritation due to the disinfecting agent in the water. Open sea bathing is an entirely different question. It is easy to see how surface infection of the water might occur on crowded beaches to a degree which might render possible transmission of infections from one swimmer to another; but quite apart from any such possibilities there is ample reason to believe that otitis or sinusitis may arise by the simple process of the washing back of infected nasal secretions into other parts of the air passages previously intact.

H. Marshall Taylor in a presidential address to the Section of Laryngology, Otology and Rhinology at a meeting of the American Medical Association this year points out that the correct approach to any medical problem is through the study of biology, and that considerations of this kind may help considerably in evaluation of facts concerning immunity and resistance.[1] He remarks that the only ætiological factor which has been at all extensively considered in the discussion of otitis and sinusitis in swimmers has been that of contaminated water, whereas the fundamental poverty of man's structural adaptations to an aquatic environment has been largely ignored. He has observed that a number of his patients with infections of the ear or sinuses have frequented a swimming pool fed by a spring having a flow of over 145,000 gallons a minute and others have not bathed except in a pool with a flow of over 365,000 gallons a minute. Repeated examination of the water of these pools has revealed low bacterial counts and for this reason no chemical disinfection has been used, so that the elements of chemical irritation and high bacterial contamination were not significant. Further, since these patients have bathed in relatively pure spring water of suitable temperature it would seem that the mechanical factor deserved consideration. This conclusion is comparable with that arrived at in regard to surf bathers of Australian beaches. Taylor states that as early as 400 B.C. the Chinese were using both chemical and filtration methods in public baths, and at the present day it is claimed by modern sanitary engineers that 99% of the bacteria in a pool can be killed or neutralized. It is further considered that if 800 gallons of water can be supplied per bather, a pool can readily be kept in satisfactory sanitary condition. Turning to biological considerations, Taylor points out that the difference between the upper air passages of man and those of the aquatic animals does not lie in any difference in the mucous membrane lining them, but in the special ability of these animals to close off the anterior nares from the water. For example, in the alligator muscular action closes the nares when the head is submerged. In aquatic birds there is also a protective mechanism varying with the species, and when water does enter the nares, it comes into contact only with a part which is not lined by respiratory mucous membranes. In the hippopotamus the nostrils are also closed on submersion, and seals and some other similar animals can close the external ear against the entrance of water. Adaptations to an aquatic existence are, of course, very much more extensive in such animals than mere details of their nasal arrangements, but, as Taylor points out, it is also very important to remember that man is strikingly at a disadvantage in other regards also. For instance, the seal maintains a normal temperature of 101° F. whether it is swimming or at rest on an ice floe, and some cetaceans can maintain a normal temperature of 104° F. even in Arctic waters. Even the stoutest surf bather must here admit man's

[1] *The Journal of the American Medical Association*, September 2, 1939.

inferiority, for man's loss of body heat during a period of submersion of twenty minutes at a temperature of 70° F. may rise to five times the normal basal rate. Nor do the human swimmers' handicaps end even here, for the delicate dermis of the external auditory canal undergoes just the same changes when immersed in water as can be seen in the epithelium of the hand when immersed in water for twenty or thirty minutes. Of course the ear of man may be artificially sealed with an impervious plug, but without such a precaution the maceration of the skin lining the external auditory canal may predispose to local infection. But Taylor believes that infections of the ear or of the sinuses may not only arise through the entrance of foreign bacteria, but also through the dissemination and multiplication of bacteria commonly present in the passages owing to a lowered resistance on the part of the swimmer. Chilling of the exterior of the body may easily be demonstrated in a group of swimmers as evidenced by the purpling of the skin, the lips and the nail beds, and even in trained athletes there may be a drop of temperature up to 4° F.

Observations on the blood and blood pressure have produced further evidence of peripheral vasoconstriction and anoxæmia. It must be conceded that ischæmia of the upper respiratory mucous membrane would be likely to lower resistance and favour infection. Of course Taylor would not decry swimming as an exercise or claim that a mere mild chilling of the skin is harmful; but he is surely right when he states that the bather who is inactive on a windy beach invites the same degree of rapid heat loss and incidentally the same risk as he would if he wrapped himself in a wet blanket and sat on a cold concrete floor. The essence of his argument is that a study of biology shows that man is conspicuously lacking in the adaptations necessary for an aquatic life, and that when he temporarily abandons that terrestrial element for which he was made he must take heed of the limitations Nature has placed upon him. It is no doubt difficult to avoid the entry of water into the upper air passages, but at least its forcible entry should be discouraged. If a positive air pressure can be maintained in the nasal cavities during submersion of the head the sinuses and Eustachian tubes will be protected, and Taylor recommends that appropriate methods of breathing be more widely used with this end in view.

TOBACCO AND OTHER DRUGS.

"If anybody would only contrive some kind of lever that one could thrust in among the works of this horrid automaton and check them, or alter their rate of going, what would the world give for the discovery?"

"From half a dime to a dime, according to the style of the place and the quality of the liquor", said the young fellow whom they call John.

If Oliver Wendell Holmes were living now he would use the modern terminology of physiology and point out that the autonomic nervous system is in normal life completely detached from voluntary control and that the use of drugs, from the innocent caffeine to the dangerous narcotics, can be explained in large measure as efforts to induce changes at will in the action of organs innervated by the sympathetic and parasympathetic systems. "Men are very apt to try to get at the machine by some indirect system of leverage or other. They clap on the brakes with opium; they change the maddening monotony of the rhythm by means of fermented liquors"—to quote again the genial autocrat of the breakfast table. The suggestion may be hazarded that there was some element of pure chance in the rise of tobacco to its immense vogue today. There is little euphoria except in the return to approximate normality in the addict when he indulges; and the first attempts of the small boy to smoke like a grown-up have effects far from pleasurable. That much of the enjoyment of smoking is purely psychic in contemplation of the smoke is evidenced by the fact that very few totally blind persons find any enjoyment in pipe, cigarette or cigar. But the American Indian who gave the world tobacco also discovered the true euphoria obtained when the leaves of the coca plant are chewed, and this habit might well have swept round the world with disastrous effects. Another drug also of American Indian origin is mescal or peyote, the use of which has spread alarmingly amongst the Red Indians of the United States of America. Some American anthropologists condone the importation from Mexico of this deliriant by asserting that it removes or relieves the inferiority complex which has more deadly effects on native populations than disease. *Cannabis indica*, which for some years has given grave concern to South Africans on account of the growing vogue amongst natives and "coloured folk", has invaded North America and entrenched itself not so much amongst negroes and Indians as amongst whites. Last year the literature on this menace was enriched by a volume of 223 pages emanating from the publishing firm of Lippincott.[1]

That the Mexican Indian has still some potent drugs which civilization may yet unfortunately adopt has been recently demonstrated by Dr. C. G. Santesson, the distinguished Stockholm pharmacologist. "Piule",[2] the seed of a convolvulus; the seed of a leguminous plant, *Rynchosia phaseoloides*;[3] "teonanacatl"[4] and "hallimahl",[5] both toadstools; and finally the seed of a malvaceous Mexican plant[5] have been investigated in the Swedish laboratory. In laboratory animals a condition of "half narcosis" is produced, but in the human hallucinations and delirium are manifested. We may indeed be thankful that with all these dire possibilities tobacco has been chosen by humanity in general from the American Indians' medicine chest as the main lever which can disturb the placid functioning of the autonomic nervous system.

[1] Robert P. Walton: "Marihuana, America's New Drug Problem: A Sociologic Question with its Basic Explanation Dependent on Biologic and Medical Principles", 1938.
[2] C. G. Santesson: "Ethnological Studies", Volume IV, 1937, page 1.
[3] C. G. Santesson: "Ethnological Studies", Volume VI, 1938, page 179.
[4] Victor A. Reko: *Magische Gifte, "Rausch- und Betäubungsmittel der Neuen Welt"*, Second Edition, 1938.
[5] C. G. Santesson: *"Einige Mexicanische Rauschdrogen", Arkiv för Botanik*, Volume XXIX, A, Number 12, 1939.

Abstracts from Current Medical Literature.

MEDICINE.

Aspiration of the Liver.

EDGAR BARRON (*Archives of Internal Medicine*, February, 1939) performed aspiration of the liver for the removal of biopsy material from 35 patients. The majority showed either hepatomegaly or jaundice. The site of aspiration was just below the costal margin, to the right of the epigastrium. The patient was given a dose of a strong hypnotic, and local anæsthesia was induced before the insertion of a number 13 gauge needle pointed in the direction of the lower border of the right axilla. When the liver was entered, continuous suction was exerted by a 20 cubic centimetre syringe so as to obtain a core of liver tissue, which was then expressed from the needle into a 1 in 10 solution of formaldehyde for fixation and microscopic section. There were no complications in the series reported, although the author mentions the possibility of hæmorrhage and the introduction of infection either from outside or from the perforation of a hollow viscus; he records the presence of colonic epithelium in one of his sections, and adds a footnote that a patient not in the series died as a result of hæmorrhage. The cases reported suggested that the procedure had been of assistance in the diagnosis, although in those in which coarse nodular cirrhosis was present the specimens obtained did not contain much fibrous tissue, and the author felt that such a finding would be unlikely by this method. It was also thought that small metastatic growths might be missed, although the diagnosis of malignant disease was made in 12 instances. Two obscure conditions were diagnosed by means of aspiration, namely, schistomiasis and congenital cystic disease, and in a case in which aspiration yielded only venous blood the patient was later proved to have a cavernous hæmangioma of the left lobe of the liver.

Anthracosilicosis.

J. F. GIEBING AND R. CHARR (*The Journal of the American Medical Association*, August 12, 1939) discuss the heart in anthracosilicosis. Previous reports have indicated that anthracite miners have heart disease more often than non-miners. Twenty-five miners were examined in this study, clinically, radiographically and by electrocardiograph. All these miners had moderate to advanced anthracosis; their ages varied between 36 and 67 years, and they had worked for from three to four years in the mines. X ray examination revealed no cardiac enlargement, except in two cases. The electrocardiograph showed evidence of myocardial degeneration in all except three. All the miners were short of breath; it was difficult to decide whether this was of pulmonary or cardiac origin. All had vital capacity reduced by 20% to 70%. Exercise increased the pulse rate and caused dyspnœa. It was noted that whereas right ventricular preponderance was expected, it was noted in only three cases; left preponderance was present in ten cases.

Polycythæmia.

PAUL REZNIKOFF (*Bulletin of the New York Academy of Medicine*, May, 1939) contributes an original article in which he discusses the classification, ætiology, incidence, pathology, symptomatology, laboratory findings and the therapy of polycythæmia. The author classifies the disorder into the mild type called erythrocytosis, similar to leucocytosis, and the pronounced type or erythræmia, analogous to leuchæmia. He draws attention to the many causes that have been advanced to explain the ætiology of erythræmia, including the hereditary and endocrine factors, the neoplastic theory and the conception of over-secretion of a gastric substance called addisin; but his own studies of the histology of the bone marrow in sufferers has led him to propose the theory that erythræmia may be due to local anoxæmia in the bone marrow itself, with over-compensation of erythrogenesis, and in any case he states that all workers are agreed that the increase of erythrocytes is not due to decreased destruction or prolonged longevity of the red blood cells, but is primarily dependent upon an increased production. The author indicates the particular occurrence of the disease in patients of Jewish origin born in eastern Europe. He describes the chief pathological changes found at autopsy as engorged vessels, hæmorrhages, thromboses, enlargement and hyperæmia of the spleen and liver, engorged marrow of the long bones characterized by erythroblastosis and leucoblastosis and an increase of the megakaryocytes. The author discusses the symptoms of polycythæmia, emphasizing those related to the nervous system; and he describes the usual laboratory findings with reference to the blood picture, the increased clot retraction time, the increased viscosity of the blood, the decreased sedimentation rate of the blood and the marked increase of the total red blood cell mass. In reference to the frequently described relationship of polycythæmia to leuchæmia, the author states that the leucogenic and thrombocytogenic tissue of the bone marrow certainly shows evidence of stimulation in most cases of polycythæmia. The paper concludes with a review of the therapy of the disease, including the use of venesection, the administration of acetylphenylhydrazine and Fowler's solution, and irradiation. The author advises the avoidance of putting polycythæmic patients to bed, attention to the emotional factors by the physician, a diet which excludes condiments, too much roughage, alcohol, hot foods and liquids which may irritate the already congested gastro-intestinal mucosa; he regards it as most important to keep the patient's blood count at a level which gives the greatest comfort and least complaint rather than at some arbitrary level suggested from a chart.

Fluoroscopy and Myocardial Infarction.

ARTHUR M. MASTER (*Journal of the Mount Sinai Hospital, New York*, May-June, 1939) discusses the fluoroscopic diagnosis of myocardial infarction following coronary occlusion, and records the observation that 66% of his series of 100 cases showed positive evidence of myocardial infarction by the use of this procedure. The author states that fluoroscopy enables the observer to see an abnormal ventricular movement in the involved region, which may assume the type of "paradoxical" pulsation or reversal of pulsation, or the type of absent or diminished pulsation. The procedure adopted by the author in the visualization of the details of ventricular contraction is to allow sufficient time for the eyes to become accommodated to darkness, after which the patient is instructed to hold in a deep breath; then systole and diastole are timed correctly by comparison of the movements of the aorta with those of the ventricle, after which the opening of the screen is diminished and the ventricular border alone is viewed. From the study of his series of patients the author concludes that reversal of pulsation in the ventricle over the affected area is characteristic of cardiac infarction due to coronary artery occlusion, that these abnormalities of contraction may be observed a few days after occlusion as well as years later, that the common sites of abnormal pulsation are the apical and subapical portions of the left ventricle, and that fluoroscopy may provide positive evidence of cardiac infarction when the electrocardiogram and physical signs are "negative".

Hypertension.

PROFESSOR RISER, J. PLANQUES AND P. BARBIER (*La Presse Médicale*, April 29, 1939) discuss cranial hypertension among arterial hypertensives. The results of 5,000 lumbar punctures showed that the normal pressure of the cerebro-spinal fluid was 15 to 20 centimetres of water in a calm, relaxed, immobile subject in the horizontal lateral posture. When the subject was in the sitting posture a pressure of 35 to 40 centimetres of water was normal; a pressure of 25 to 60 centimetres indicated mild cranial hypertension; one of 40 to 80 centimetres severe hypertension. In 140 arterial hypertensives it was recorded that uncomplicated arterial hypertension did not determine cranial hypertension. However, when defective hepato-renal function or cardiovascular complications occurred, the cranial tension was increased. Among 50 such patients the cerebro-spinal fluid pressure was between 23 and 53 centimetres of water when they were

in the horizontal position. Headache and mental confusion were often noted with normal intracranial pressure, but frequently indicated an increased pressure. The chief retinal sign of intracranial hypertension was papillœdema (70% of cases recorded). Venous pressure was always raised above 15 centimetres in cranial hypertension, though the blood urea was not necessarily increased. The chlorides in the spinal fluid were often increased. Œdema of the meninges with serous meningitis also occurred at times. Persistent cranial hypertension was a definite sign of malignant hypertension.

Vitamin B_3.

J. V. LANDOR (*The Lancet*, June 17, 1939) discusses deficiency of vitamin B_3, nicotinic acid. Black tongue in dogs and human pellagra have been cured with this preparation. Stomatitis, diarrhœa and lesions of the mucosæ have been cured also. In Malaya a deficiency disease occurs in prisons, with eczema of the scrotum, angular stomatitis or sore tongue, curable by yeast or "Marmite". In August, 1938, ten prisoners suffering in this way were treated with 0·5 gramme of nicotinic acid given daily for fourteen days. They did not improve. Half an ounce of "Marmite" was then given daily to each patient. The result was rapid improvement in every instance. The scrotum was examined for fungus in all cases, and in only one was mycelium found. Two prisoners with angular stomatitis were treated with smaller doses of nicotinic acid, 0·15 gramme daily. Each patient had scrotal eczema also. There was no improvement. It appears that eczema of the scrotum and stomatitis in Singapore jails are not cured by nicotinic acid. The author suggests that these lesions may be a different type of pellagra and that there may be other factors which prevent pellagra besides nicotinic acid.

Occlusive Arterial Disease of the Legs.

NELSON W. BARKER (*Annals of Internal Medicine*, May, 1939) reports two cases of occlusive arterial disease of the legs associated with lipæmia and *xanthoma tuberosum*. Both patients were in the fifth decade of life, and complained of typical intermittent claudication of the calves of both legs; they had typical nodular yellowish cutaneous lesions in the region of the elbows and knees. Pulsation was absent or diminished in the *dorsalis pedis*, posterior tibial and popliteal arteries, but there was no evidence or history of phlebitis, pain or trophic changes in the toes, or symptoms referable to the upper extremities. The general physical examination gave negative results, but the value for blood cholesterol was 667 milligrammes *per centum* in one case and 657 milligrammes in the other. There was also an increase of the values for the fatty acid and total lipoid content of the blood. A diet low in fat and cholesterol content was used in treatment, but the defect in pulsations remained unaffected, whereas the intermittent claudication was alleviated in the one case and not in the other. The concentration of lipoids in the blood was reduced, but not to a level within normal limits. Diets containing thirty grammes of animal fat a day were well tolerated, but an attempt to give these patients diets containing less than two grammes of animal fat a day was abandoned owing to increasing muscular weakness and fatigue. The author draws attention to the possible relationship between the hyperlipæmia and hypercholesterolæmia and the occlusive arterial disease manifest in his two cases. The high concentration of the blood lipoid, the age of the patients and the absence of superficial phlebitis or involvement of the arteries of the upper extremities lead the author to the conclusion that the most likely possibility is that the arterial lesions were premature extensive atheromatous formation, rather than the result of thrombo-angiitis or arteriosclerotic *endarteritis obliterans*. He emphasizes Montgomery's statement that peripheral arterial disease may occur in association with *xanthoma tuberosum*, and as this type of xanthomatosis may be asymptomatic, patients with peripheral arterial disease should be examined for evidence of xanthomatous lesions and patients with xanthomatous lesions should be examined for evidence of peripheral arterial lesions.

"Sedormid."

T. MCGOVERN AND I. WRIGHT (*The Journal of the American Medical Association*, April 29, 1939) reported a case of *purpura hæmorrhagica* after the use of "Sedormid" (allyl-isopropyl-acetyl-carbamide). Thirty-seven cases of purpura due to "Sedormid" have been reported. In the authors' case a woman, aged fifty-seven years, took one half tablet of "Sedormid" every night for several months with no ill effects. After a lapse of one month she took one tablet the night before the onset of purpura. She became acutely ill, with headache, chills, fever and thirst, and found her skin spotted with hæmorrhages. Two months later she again took one half tablet of "Sedormid"; within two hours she felt chills and fever; next day the mucosa of her mouth was covered with hæmorrhages and the skin with petechiæ and ecchymoses, which took three weeks to disappear.

Values and Limitations of Electrocardiography.

HERMAN TARNOWER (*New York State Journal of Medicine*, June 1, 1939) discusses the values and limitations of electrocardiography, and states that the electrocardiograph is the most precise means at our disposal for investigation of the state of the myocardium, although the degree of abnormality in the tracings does not always parallel the amount of cardiac damage in so far as it depends not only on the amount of damage but also on the site. The author is of the opinion that electrocardiography should be employed with thoughtful discrimination, rather than as a routine procedure, that its use is indicated when a diagnostic question remains after the clinical features of a case have been thoroughly studied, and that a careful correlation of the clinical data is essential for the satisfactory interpretation of an electrocardiogram. The author states that an electrocardiogram may be of great aid in the diagnosis and management of cases of myocardial infarction, acute rheumatic fever, congenital heart disease, constrictive pericarditis, and the various cardiac arrhythmias, but has a very limited value in other diseases of the heart. In his opinion, the fourth lead can be advantageously employed when myocardial infarction is suspected, but the standard leads alone will suffice for all other conditions.

Peptic Ulcer.

H. A. WARREN, M. PIJOAN AND E. S. EMERY (*The New England Journal of Medicine*, June 29, 1939) discuss ascorbic acid requirements in patients with peptic ulcer. Normal persons utilize from 0·4 to 0·8 milligramme of vitamin *C* per kilogram of body weight per day. When saturation exists any excess of vitamin *C* is excreted in the urine. Five patients with duodenal ulcer were studied. They had been on diets presumably low in vitamin *C* for one to seven weeks before admission to hospital. Necessary daily requirements to maintain saturation were approximately one milligramme per kilogram of body weight. This was 20% greater than has been found for normal subjects. Patients with peptic ulcer therefore used more ascorbic acid than normals. The amount was small, however, and could be supplied by the juice of one to two oranges. Sippy diet in the first week contained five milligrammes of ascorbic acid per day; in the fourth week, 15 milligrammes. The normal requirements were 70 milligrammes per day. However, there was no evidence that patients on Sippy diet suffered any ill effects from this deficiency of vitamin *C*, nor was there any evidence that their ulcers did not heal satisfactorily on Sippy diet. The need for the addition of vitamin *C* preparations or foods is therefore at present quite theoretical. If the physician wishes to prescribe an adequate amount of vitamin *C*, this can be done by the giving of 200 milligrammes of ascorbic acid per day for one or two weeks, then 75 milligrammes per day for two or three weeks longer. At the end of that time the patient should be able to obtain an adequate amount from his diet. Orange juice, tomato juice or crystalline ascorbic acid can be used. There was no evidence in this study that patients with peptic ulcer could not absorb ascorbic acid given by mouth.

Special Articles on Civilian War Casualties.

V.

THE TREATMENT OF WAR WOUNDS.

In the short time at my disposal it will be impossible to do more than give an introduction to the subject of wounds and their treatment. I shall endeavour to state the principles on which treatment is based, and to indicate the application of the knowledge gained in the treatment of war wounds to those encountered in civil practice.

It is only right that our methods of dealing with casualties in any emergency should be governed by our experience in previous wars; but there is a danger that our methods of treatment may become stereotyped and standardized and so put into practice in any emergency without recognition of the fact that the conditions with which we have to deal have changed. As an example of what I mean I shall refer to the experience gained in the Boer War. In this war most of the wounds were caused by high velocity rifle bullets fired at long range. Such bullets were sterile on striking their objective, and the only sources of infection were the clothes and the skin of the soldier; further, the bullet as a rule produced a clean penetrating wound with a minimum of damage to the tissues, and very often was not retained in the body. Also the army medical services were in a primitive state of development compared with their thorough and complete organization in the Great War and at the present time, so that in the Boer War surgical aid was inevitably delayed and was often of poor quality. It was found by experience that the victims of these wounds did well without operative interference, that wounds of the thorax almost always had a favourable course and that even penetrating abdominal wounds were not necessarily fatal.

As a result of this experience conservative methods of treatment were accepted as sound in principle, and it was with these ideas that the medical services entered on the Great War. The disillusionment was rapid and complete. Within the first week or two of fighting it was realized that the damage to tissue and organs was severe, that almost every wound became septic, that the incidence of tetanus and gas gangrene infection was very high, and that the mortality was appalling. The difference in the two cases is apparent; in Flanders the majority of wounds were due to high explosive shells, which burst in the ground into irregular fragments and carried into the tissues grossly infected dirt and clothing; the damage to muscles and organs was very great, and most of the shell fragments were retained in the body. Even shrapnel balls and rifle and machine-gun bullets might introduce infection from dirty clothing and skin.

Before very long the medical services became organized so efficiently that skilled surgical treatment was available in the great majority of cases within a few hours of the soldier's being wounded. For these reasons the accepted treatment of gunshot wounds was radically altered, and it was laid down that every wound must be operated on without delay (within a few hours if possible) and the foreign body, if present, removed.

Professor MacCallum has described the nature and variety of the wounds inflicted in the Great War. He has shown how they range from tiny penetrating wounds, so small that they may be overlooked, to extensive widely open wounds with great destruction of tissue. He has demonstrated the fact that a tiny fragment with a trivial entrance wound can cause very great and even fatal internal injuries. He has demonstrated the injuries to viscera, blood vessels and nerves, and has described the infections that may be present.

I shall now discuss the treatment of such wounds, and I should like to stress the principles on which the treatment is founded rather than the technique (for the latter will change with increasing knowledge and varying conditions) and also the application of these principles to civil practice.

First aid treatment consists merely in the stopping of hæmorrhage by the application of a tourniquet or by pressure (probably no other method will be available), in the application of a simple dressing to the wound and in the prevention of shock. I shall go into this last question, namely, the problem of shock, in some detail. It is, of course, obvious that bleeding must be controlled and that pain must be relieved by large doses of morphine. One-sixth to one-quarter of a grain of morphine is useless; one-half to one grain is often necessary before pain is relieved. Immobilization of a severely damaged limb is essential, even if no fracture is present. An open wound of the thorax must be closed by a large dressing, so that air does not enter and leave the thorax with respiration. Lastly, the question of warmth is all-important. First aid posts should be equipped so that patients can be made warm and comfortable, and motor ambulances should be heated in cold weather.

The next thing to do is to get the patients to a hospital where radical surgical treatment is available without delay. In France during the Great War this was often accomplished within five or six hours of the soldier's being wounded; in cities, in the case of wounds caused by accidents or bombing from the air, the patient should be in hospital within two hours. This question of speed is all-important; delay inevitably means increase in the shock and progress of the septic infection which is always present. I would stress the fact that delay of several hours may be decisive as to the wound's healing with a minimum of infection and little or no permanent disability on the one hand, or on the other hand the development of severe sepsis, perhaps tetanus or gas gangrene, a long severe illness, and probably permanent disability or death. The delay which occurs in civil hospitals in the treatment of accident casualties is often inexcusable.

On arrival at hospital the patient should be operated on without delay. The only reason for not operating at once is the presence of shock, which may be so profound as to render immediate operation dangerous. There is no doubt that civilians will be affected much more gravely in this respect than soldiers. This was the experience in Barcelona as the result of air raids, and must have been observed by any surgeon who has had to deal with a large-scale railway accident. It would appear that terror plays a large part in producing shock, and this is a factor which would be encountered to a maximum degree in air raids on cities.

The treatment of shock includes administration of blood transfusions, and will be dealt with in detail in a subsequent lecture.

We now have to consider the operation. Every victim of shell or bomb wound, no matter how trivial the wound may appear, should be operated on. Operation may be omitted in cases of wounds by rifle bullet and perhaps by shrapnel ball, provided there are no hæmorrhage and no injury to tissues or organs which require repair.

The conditions to be dealt with at operation are the following:

1. Control of bleeding by ligature of all bleeding vessels. Extravasated blood predisposes to the development and spread of infections, notably anaerobic infections. When main arteries, such as the femoral or brachial, have to be ligated in their course, it has been stated with some authority that the risk of gangrene is lessened if the accompanying vein is also ligated. I am unable from my own experience to confirm this statement, and I am inclined to doubt its truth.

2. Prevention of sepsis. It must be assumed that every shell wound is infected. The surrounding skin is first sterilized, then the wound in the skin and subcutaneous tissues is excised. If the wound is not very extensive the whole of the surface of the wound may be excised in one piece. The wound is then opened up to its whole extent; no recesses which may harbour infection are left; all dead and damaged tissue is cut away. The foreign body, with the dirt and clothing surrounding it, is removed; this is

[1] A lecture delivered under the auspices of the Melbourne Permanent Post-Graduate Committee on July 24, 1939.

an essential point of the operation if sepsis is to be prevented. The wound may then be washed out carefully with flavine solution or eusol, every part being cleansed or treated with spirits and bismuth-iodoform-paraffin paste after the method of Rutherford Morrison.

Drainage, free and continuous, is the most effective means of combating infection, and antiseptics play a secondary part. The wound is left wide open; no sutures are put in or, alternatively, sutures may be inserted but are not tied till several days later, when the danger of infection is passed. I regard this point of not suturing the wound as of the greatest importance in the treatment of civil injuries which we are called on to deal with every day. It must be the experience of everyone that wounds which are the result of motor car and machinery accidents, if sutured, suppurate freely, and the sutures have to be removed in the course of a week or so. Such wounds should never be sutured except perhaps in the scalp or face.

I would also stress the fact that drainage by a rubber tube is not sufficient; further, it may be dangerous, in that it causes in many cases erosion of arteries and severe hæmorrhage.

I have said that the foreign body should be removed in every case provided it is a piece of shell or bomb. This does not apply to the bullet if it is retained, and the practice of "operating to remove the bullet", so common in civil practice, is to be deprecated. It is in many cases doing no harm and is much better left alone.

3. Repair of the damage. The third object in operating is to repair the damage that has been caused. In the case of visceral injury this is imperative and urgent. Other tissues, such as nerves and tendons, should not be repaired till several days later, when the danger of sepsis is past and the wound can be sutured.

Finally, I would remind you of the beneficial effect on an infected wound of rest and immobilization. Judicious splinting will be beneficial to all severe wounds, and thorough immobilization will play an important part in the control of infection in compound fractures.

I might mention here the question of X ray examination before operation. In the large majority of cases this is not necessary, as the fragments of shell can be felt in the depths of the wound. Moreover, some fragments are aluminium and so are not shown by X rays. In some cases it is important to know the course of the fragment so that a diagnosis as to what organs are injured may be made. This can be done by a consideration of the wound of entry and of the position of the foreign body as shown radiologically.

We now have to consider special infections, and the two that concern us are tetanus and gas gangrene due to anaerobes.

As regards the prevention of tetanus, the treatment of this disease hardly comes within the scope of this lecture. Every victim of a penetrating gunshot, shell or bomb wound should receive a prophylactic injection of tetanus antiserum within a few hours of the infliction of the wound. This also applies to wounds received in the street or country, but does not apply to abrasion and cuts from clean knives which could not possibly be infected with the tetanus bacillus. I think that this injection is often made unnecessarily, and occasionally it is harmful.

The anaerobic infections which cause gas gangrene are of the utmost importance. Several different organisms are concerned in this disease, so it is obvious that no reliance can be placed on serum therapy. The organisms are present at first as saprophytes in extravasated blood and damaged tissue. Later they invade the muscles, and this invasion is accelerated and enhanced by the presence of other organisms, especially hæmolytic streptococci, and by dirt. At this stage the infection is purely a local one and is confined to a particular muscle or group of muscles. The patient suffers from severe toxæmia. Finally the organisms enter the blood and a gas septicæmia develops; this event is followed in a short time by the death of the patient.

The symptoms and signs of the disease should be recognized by every medical practitioner, as any delay in treatment may be fatal. The patient is pale and in a state of profound shock, the pulse is rapid, and he complains of very great pain in the wounded part. There is a discharge, not necessarily copious or purulent, with a characteristic fæcal odour; the surrounding skin is bluish in colour, the tissues are swollen and may be crepitant. Bubbles of gas may be expressed from the wound, and X ray examination reveals lines of gas in the intermuscular planes.

Safety lies only in immediate operation. The wound is excised and an incision is made the whole length of the affected area. There must be no hesitation in making the incision of adequate length, even if it goes the whole length of the limb. The infected muscles are exposed; in the early stage they are swollen and coagulated and have lost the power of contracting; later they become black and diffluent. All the diseased muscles must be completely excised, and if such apparently ruthless destruction shocks the surgical feelings of the surgeon he can console himself with the knowledge that the muscles are doomed to destruction in any case. The wound is then washed out with hydrogen peroxide or eusol and is left widely open. If the excision has been complete the patient will recover and the wound may be sutured in the course of one or two weeks.

In some cases of compound fractures and in those in which there has been delay in proper treatment, the infection may be so widespread that no operation short of amputation can save the patient's life.

Anaerobic infection is also encountered in gunshot wounds of the thorax. In this case the disease starts as an infected hæmothorax, and later the organisms invade the chest wall and the lung. By early operation and clearing out of the infected clot cure can be confidently expected.

When the wound involves a joint the wound is thoroughly excised, as previously described, and the joint is widely opened. The foreign body and any loose fragment of bone are removed and the joint cavity is washed out with acriflavine solution. The synovial membrane is then sutured and the cavity closed completely. The superficial tissues—capsule, aponeurosis and skin—are left widely open, and a secondary suture is performed several days later, when the danger of infection is past.

A limb may be so badly damaged that amputation is inevitable. Also amputation may be necessary in severe cases of infection or gas gangrene. In all such cases a guillotine amputation is the operation of choice, the limb being removed either through the destroyed area or as close to it as is safe, or through the joint immediately above the injured or infected area. Later, when infection is eradicated, the final amputation can be performed. If a set amputation with flaps is performed as a primary operation infection and breaking down of the wound almost inariably follow, and the result is prolonged sepsis, further loss of tissue and probably a reamputation at a higher level. The guillotine amputation provides that drainage which is necessary to cure infection, it prevents perhaps months of chronic sepsis, and it finally saves tissue and provides the maximum length of limb which is possible.

C. GORDON SHAW, D.S.O., V.D., M.D., F.R.C.S., F.R.A.C.S., Colonel, A.A.M.C.

Melbourne.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on August 31, 1939, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. G. M. BARRON, the President, in the chair.

Steatorrhœa.

DR. LAURENCE HUGHES read a paper entitled "Steatorrhœa" (see page 671).

DR. F. S. HANSMAN read a paper entitled "Steatorrhœa" (see page 677).

DR. MARGARET HARPER said that congenital steatorrhœa due to pancreatic defect had been described by Garrod and Huntley in 1913. They reported the condition in two brothers, one of whom died when eleven months old; no *post mortem* examination was performed. The other was alive and working when fifteen years of age. Since that time isolated cases of congenital steatorrhœa with *post mortem* examinations had been reported, mainly in England and Germany. Dr. Harper went on to say that she had reported her first two cases in 1930; in one of the reports autopsy findings were included. It was only in recent years that the clinical condition of congenital steatorrhœa had been linked up with a pathological condition of the pancreas. Clarke and Hadfield in 1924 had been the first to report a case with the results of *post mortem* examination and to distinguish this condition clearly from cœliac disease. Dr. Harper's own series of cases included those of fourteen children. Two of these were still alive at the ages of twenty months and three and a half years. The condition of all these children had been diagnosed as congenital steatorrhœa due to a defect in the pancreas; and the diagnosis had been confirmed *post mortem* in six cases in which autopsies were performed. In all of these the condition of the pancreas had been identical. Small distorted acini with shrunken cells were present, the ducts were dilated and tortuous, fibrous tissue was increased, but the islets of Langerhans were normal in appearance. The picture suggested that the gland substance was immature and that this immaturity was followed by fibroid hyperplasia. There was no evidence of inflammatory reaction. In one of Dr. Harper's cases and in four others that had been reported the acinar tissue was entirely replaced by fat.

Symptoms were present from birth. In one case, in which the infant was under observation from the second day of life, the diagnosis was made in the third week. A failure to gain in weight on a diet normal in quantity and quality and the peculiarity of the stools were the presenting signs. The stools were frequent and bulky and had a pale green or creamy and lustrous colour; they were known as "butter stools" and had a rancid odour. Microscopic examination of the stools revealed numerous fat globules with few or no soap crystals. The percentage of fat in the dried fæces was high, and in this fat the percentage of unsplit fat was usually, though not always, high. When the patient was on a low fat diet the percentage of unsplit fat was usually lower than when a full fat diet was given. At this stage Dr. Harper showed a glucose tolerance curve typical of the condition. She said that practically all the infants reported had died of bronchopneumonia. All of her patients except one had died of this complication. Quite early in the illness a persistent spasmodic cough might develop, and this was frequently mistaken for pertussis. This cough might last for some months before the terminal bronchopneumonia developed. The reason for this susceptibility to respiratory infection was not clear, though one naturally suspected that it might be due to a failure in the utilization of vitamin *A*.

Dr. Harper said that Dorothy Anderson had reported that in 10 of 44 patients in whom the pancreatic lesion was present there was found evidence of metaplasia of the epithelium in lungs, kidneys and other organs, such as was usually caused by vitamin *A* deficiency. In two of Anderson's cases xerophthalmia had been present during life. Blackfan and Wolhbach in 1933, in searching for evidence of vitamin *A* deficiency in *post mortem* material of the previous ten years, found evidence of vitamin *A* deficiency in eleven instances; in seven there were found "extensive pancreatic lesions, all identical and presumably representing a disease entity". This report had been made before the connexion between congenital steatorrhœa and the pancreatic lesion was recognized in the United States of America. The description of these lesions was exactly the same as that of the lesions found in Dr. Harper's cases and in others that had been reported. Dr. Tidswell had been unable to find metaplasia of the epithelium in Dr. Harper's cases, and no xerophthalmia was found.

Lindquist, in a recent review of the relation of disease to vitamin *A* deficiency, had reported experiments to determine the most satisfactory method of measuring the carotene and vitamin *A* content of the blood serum. He determined the range of vitamin *A* values in the blood serum of normal people and he concluded that less than 70 international units per 100 cubic centimetres of blood serum indicated a serious shortage, that 70 to 100 units indicated a low content, that 100 to 200 units were apparently sufficient for many people, and that 200 to 400 was the amount that might be considered really satisfactory. Ranch and others had recently suggested that there was in these cases a congenital anomaly of development of the lungs as well as of the pancreas, and that this was the reason why all these children died of bronchopneumonia. They quoted Fanconi and his associates as being of the opinion that bronchiectasis with malformation of the pancreas and chronic nutritional insufficiency was both congenital and hereditary. If lack of vitamin *A* was the cause of bronchopneumonia, why, Dr. Harper asked, was the evidence of this lack not found more frequently? Why was there not the same susceptibility to respiratory infections in cœliac disease?

In regard to the familial aspect of the condition, Dr. Harper said that two of her patients had siblings who died of the disease. In another instance three siblings died of bronchopneumonia with disturbance of nutrition. In one instance the disease had been reported as being present in twins, and Burghard had reported an instance in which one twin suffered from the disease and the other was healthy. Occasionally other congenital anomalies had been present. Three of Anderson's patients had died in the neonatal period from atresia of the intestine; three of Dr. Harper's patients had siblings who died of developmental defects of the alimentary tract.

Discussing treatment, Dr. Harper referred to the use of pancreatic extract and of "Tabloid" carotene, 0·002 gramme. She also referred to the bad prognosis, but said that Parmelee had reported one child who lived to be eleven and a half years old.

In conclusion Dr. Harper referred to sprue and to Véghley's *Giardia lamblia*. In regard to the former Hamilton Fairley had said: "My own view is that sprue arises as a functional breakdown in the gastro-intestine and that any factor or factors depressing its secretory or absorptive function will predispose." In regard to the latter Emerson had said: "This parasite lives in the jejunum and duodenum and sits on the top of a columnar cell which it embraces with its concavity. In some cases they are found in such numbers that they form a membrane covering the mucosa."

DR. ALLAN WALKER said that he thought that the three speakers had accepted the challenge to present the subject of steatorrhœa in an interesting way. Moreover, they had proved that there were in Sydney three people who knew something about it. Dr. Walker went on to say that his own interest lay rather in the adult type, known as idiopathic steatorrhœa of adults. He agreed with Dr. Hughes in concurring with the opinion of Thaysen that cœliac disease in children and steatorrhœa in adults were one and the same. Dr. Walker had very little personal experience of tropical sprue, but he recognized that there was some reason for regarding it as being different from the non-tropical variety of steatorrhœa. The greater frequency of glossitis and of hyperchromic anæmia indicated a difference, which might, of course, be only of type and degree. Dr. Walker was interested in Dr. Hansman's description of experiments with heavy hydrogen; but he had noted that the case in which this experimental work had been done was not one of the clinical type under discussion. He thought that it would be of value if the experiment could be carried out in cases of steatorrhœa of the so-called idiopathic type.

Dr. Walker then said that steatorrhœa in the adult was a very interesting disease, which raised some questions not easy to answer. If the idea was accepted that cœliac

disease in the child was the same, the question remained why it might occur in the young child and yet might in other cases start in adult life. This problem was, of course, a common one in the history of disease. An even more interesting problem remained. Idiopathic steatorrhœa in the adult was not seldom observed to be cyclical in nature; and remembering that it brought in its train anæmia, which occasionally was of the hyperchromic type, it was interesting to reflect that the commonest type of hyperchromic anæmia, namely, pernicious anæmia, was also cyclic, or had been until the introduction of liver therapy. It was reasonable to suppose that if the cause of the condition acted without intermission there would be no remission of symptoms; but the condition did wax and wane, though the reason for this was obscure. Another interesting point was that all the significant symptoms were of the negative or deprivation order. The loss of weight of the patients, other evidences of the non-absorption or non-utilization of fat, the presence of fat in the stool, interference with the normal utilization of sugar or, more correctly speaking, the presence of a low blood sugar curve, the presence of anæmia, the presence of changes due to disordered calcium and phosphorus metabolism, and to vitamin deficiencies, all these were deprivation symptoms. Referring to the discussion of the cause of the flat blood sugar curve, Dr. Walker felt that the endocrine disturbances mentioned by Dr. Hughes as a possible factor were likely to be much more relevant than fermentative action in the bowel. He agreed with Dr. Hansman that the latter explanation was surely incorrect. The anæmia was a curiously changeable one; in the non-tropical variety it might be hyperchromic in variety, and its type might also vary from time to time. Dr. Walker drew attention to the wide variations in the mutual relation between the red corpuscle count and the hæmoglobin value in the tables shown by Dr. Hughes. The colour index in these children showed a very wide range of difference. It seemed that the defect might not only be an iron deficiency and that there might be a possible lack of marrow stimulation or of the essential blood-forming factor. Dr. Walker remarked that it was interesting that some of the adult patients suffered from muscular cramps. These were, of course, of the nature of tetany. Apart from this, though muscular metabolism was interfered with and wasting was evident, it was of interest that the nitrogen metabolism of the body was unchanged. To the clinician one of the most striking features of idiopathic steatorrhœa of adults was that although it was a very obscure disease, which needed a great deal of investigation before its presence could be definitely proved, and called for much more research before it could be understood, yet it could be diagnosed at the bedside with reasonable certainty. The patient had a characteristic history of weakness and of waxing and waning of symptoms, the stool was characteristic, there was definite wasting of tissue, causing a striking contrast between the protuberant abdomen and the rest of the body, anæmia was present, and changes suggestive of vitamin and calcium deficiency were found. If the patient's condition was considered from the point of view of one symptom only, a mistake would very likely be made. The position was comparable with that of blood disease, which must be regarded, as Castle had pointed out, not as an affection of one tissue only, but as one concerning the body as a whole. The disorder must not be looked upon as a biochemical curiosity; it was, but it was also a clinical picture that was clearly recognizable.

DR. E. H. M. STEPHEN expressed appreciation of the manner in which the three speakers had presented the different aspects of steatorrhœa. The prognosis in congenital steatorrhœa contrasted strikingly with that in cœliac disease. The patient with cœliac disease with muscular hypotonus, abdominal distension and general passivity (with a leaning towards pessimism) was likely under suitable treatment to develop into athletic elegance, whereas in congenital steatorrhœa there was the tragic prospect of early decease after a diet of peculiar difficulty.

DR. LORIMER DODS said that he had been particularly glad to hear Dr. Hansman emphasize the fact that the estimation of the percentage of fat in a single specimen of fæces was unsatisfactory, and also the need for fat balance experiments in a complete study of steatorrhœa. Fat balance experiments were at the present time rather beyond the scope of the hospitals, but he hoped that it would be possible to arrange to have such experiments carried out later. In the absence of such detailed studies as fat balance experiments, Dr. Dods suggested that the ideal procedure to follow was that recommended by Professor Parsons, namely, the examination of a three to five day specimen of fæces and the estimation of the total daily excretion of fat in grammes. Professor Parsons insisted on a total daily excretion of more than two grammes of fat as one of the criteria for the diagnosis of cœliac disease. In the assessment of any results of fæcal fat estimations in early infancy it had to be remembered that a mild and simple diarrhœa might be responsible for a temporary failure of absorption of fats and even some failure of splitting of fats.

Dr. Dods had been glad to hear Dr. Margaret Harper's remarks on congenital steatorrhœa, as he thought that Dr. Harper had had more experience with this disorder than any other pædiatrician in the world. Dr. Dods regretted that Dr. Hughes had not had time to discuss the work of Ross, of Melbourne, on the use of liver in the treatment of cœliac disease. Ross had shown that the high "intravenous" curve of cœliac disease could be brought to normal by the administration of liver preparations, such as "Campolon", and had emphasized the value of "Campolon" and crude liver extracts in the treatment of cœliac disease.

Dr. Dods then related a story told by Professor Parsons. During the Great War cœliac disease was put on the list of diseases for which extra meat and extra protein foods could be obtained. This resulted in an epidemic of cœliac disease of such a size that its name had to be withdrawn from the lists.

Dr. Hughes, in reply to Dr. Hansman, who had criticized the theory that fermentation alone was responsible, said that he quite agreed with Dr. Hansman. He had mentioned the theory only because it was one of those put forward to explain why proper absorption failed to take place. It was interesting, however, to notice that the administration of banana did improve the condition of some patients with cœliac disease. Dr. Hughes was glad that Dr. Harper had mentioned *Giardia lamblia*. That question had been discussed in some detail by Miller during the Great War days; he had stated that *Giardia lamblia* infection was at times associated with steatorrhœa. In reply to Dr. Walker, who had referred to tropical sprue and its similarity to steatorrhœa, Dr. Hughes said that he had stressed two points. They were: (i) the fact that the anæmia in tropical sprue was of the hyperchromic type, whereas in children with cœliac disease it was hypochromic; (ii) the question of stomatitis. In his experience stomatitis was an invariable symptom in tropical sprue, but it was not a common finding in cœliac disease. It should, however, be remembered as a possible symptom.

Dr. Hansman, in reply, said that he did not wish Dr. Hughes to think that he had been criticizing Dr. Hughes's belief. Dr. Hansman had been reading Bodenock and Morris's work on steatorrhœa, and the fermentation theory had been mentioned by them. Another of their theories was that dilatation of the colon was a protective mechanism. Dr. Hansman could not accept this view. Referring to Dr. Harper's question as to whether it would be possible to devise a simple method for estimation the content of vitamin *A* in the blood, Dr. Hansman said that he had had no experience of such an estimation, but that there were several methods by which it could be carried out. It would be difficult, however, to get sufficient blood.

VICTORIAN BRANCH NEWS.

THE following information of interest to the members of the Victorian Branch of the British Medical Association is published at the request of the Council.

Infectious Diseases: Period of Exclusion from School.

The Association of Head Mistresses of Girls' Secondary Schools of Victoria has intimated that some medical practitioners are giving certificates for children to return to school, after suffering from infectious diseases, much earlier than is provided in the Infectious Diseases Regulations of 1934.

As penalties are provided for failure to observe those regulations, the First Schedule is published for the information of members.

Exclusion of Patients and Contacts from School.

(1) For the purpose of this schedule—

"Contact" means child of school age or pre-school age living in the same house as the patient.

"Isolated" where applied to a patient means isolated in a hospital or in any other place (including place of residence) to the approval of the medical officer of health.

"Patient" includes carrier.

(2) No patient or contact shall attend school unless the conditions (where applicable) hereunder prescribed are complied with.

Disease.	Patients shall be Excluded from School.	Contact shall be Excluded from School.	
		Where Patient is Isolated, or where Contact is Removed to a House where there is no Infectious Disease.	Where Patient is not Isolated.
Diphtheria	Until a medical certificate of freedom from infectivity is furnished. No such certificate shall be issued until two swabs taken at intervals of 48 hours are negative.	Until a medical certificate is furnished to the effect that they are not liable to convey infection: Provided that where there is no medical attendant the contact may attend school 14 days after the last exposure to infection if apparently in good health and free from sore throat or discharge from nose.	Until the patient has resumed or might lawfully resume school attendance: Provided that the contact is apparently in good health and free from sore throat or discharge from nose.
Scarlet fever	Until a medical certificate is furnished that he is not liable to convey infection or until all traces of peeling of the skin and discharge from the eyes and ears have disappeared; but in any case until six weeks have elapsed since the beginning of the illness.	Until ten days after last exposure to infection.	Until patient has resumed or might lawfully resume school attendance.
Measles	Until a medical certificate is furnished stating that he is not liable to convey infection or until all traces of the rash have disappeared and there is no sign of discharge from ear, eye, or nose; but in any case until four weeks have elapsed since the beginning of the illness.	Until fourteen days after last exposure to infection have elapsed.	Until patient has resumed or might lawfully resume school attendance.
		Provided that contacts who already have had measles shall not be excluded.	
Whooping cough	Until a medical certificate is produced stating that he is not liable to convey infection or until whooping has ceased for two weeks but in any case until the expiration of six weeks from the beginning of the whoop.	Until fourteen days after last exposure to infection have elapsed.	Until patient has resumed or might lawfully resume school attendance.
		Provided that contacts who already have had whooping cough shall not be excluded.	
Cerebro-spinal meningitis	Until a medical certificate is produced stating that he is not liable to convey infection.	Until twenty-one days after last exposure to infection have elapsed.	Until a medical certificate is produced stating that he is not liable to convey infection.
Polioencephalitis, poliomyelitis, and encephalitis (lethargic) ..	Until a medical certificate is produced stating that he is not liable to convey infection.	For a period of 21 days after the commencement of patient's illness.	
Trachoma, tuberculosis (pulmonary) and typhoid fever ..	Until a medical certificate is produced stating that he is not liable to convey infection.	Not to be excluded.	
Chicken pox	For two weeks or until every scab has fallen off.	Not to be excluded.	
Mumps	For three weeks after the beginning of the illness.	Not to be excluded.	
German measles	For two weeks after the beginning of illness.	Not to be excluded.	

Conditions Governing Employment of Assistants in General Practice as Approved by the Council of the Victorian Branch of the British Medical Association.

1. In every case a written agreement should be entered into between principal and assistant embodying *inter alia* the following principles:

(*a*) The assistant to promise to give diligent and faithful service and to devote to the practice of the principal such time as may be specified.

(*b*) The assistant to receive from the principal periodic payment for his services.

(*c*) The assistant to faithfully account for and pay over to the principal all moneys received on behalf of the practice.

(*d*) Provision for a holiday for the assistant.

(*e*) Provision for the duration and determination of the agreement.

(*f*) A restrictive clause governing the future right of the assistant to practise.

(*g*) Provision that both principal and assistant become and remain members of the Medical Defence Association of Victoria.

(*h*) Provision for the settlement of disputes by arbitration.

2. A whole-time assistant should be entitled to not less than:

(*a*) two weeks' holiday a year on full pay,

(*b*) two weeks' sick leave (non-cumulative) *per annum*,

(*c*) twenty-four hours' unbroken relief from duty in each week.

3. The offer or promise of a future partnership should not be made a reason for paying a lower salary to an assistant, and if an assistant is engaged on the basis of a lower rate with a view to partnership the amount by which the salary of the assistant was so reduced should be credited to him, and should the partnership not eventuate he should be paid that amount at the termination of his contract.

4. The following minimum salaries should be paid to whole-time assistants:

£8 8s. per week for the first year,

£10 10s. per week subsequently.

In all cases board and lodging and overhead expenses of practice, including transport, should be provided by the principal.

If not provided, the following additional allowances should be paid:

In respect of board and lodging, £2 2s. per week.

In respect of motor car, £2 2s. per week.

Obituary.

ALEXANDER SYDNEY JOSKE.

WE are indebted to Dr. H. Boyd Graham for the following account of the career of the late Dr. Alexander Sydney Joske.

Dr. A. S. Joske, who died suddenly on September 5, leaves a fragrant memory with those who were in any way privileged to have been associated with him, more particularly in the lighter mood. His many-sidedness was an outstanding characteristic, and people distinguished in diverse walks of life will miss him.

He was born in Sydney on January 10, 1863, but the family moved to Melbourne while he was very young. His father, Adolph Joske, a merchant, died in 1872, and the mother supported her family by teaching. In 1875 Alexander Joske entered the Melbourne Grammar School, won prizes for special prowess in history and languages, passed the matriculation examination in 1877, and stayed in the senior classes for a further two years. In 1880 he went on to the University of Melbourne and graduated in medicine at the end of 1884 and in surgery in the following April.

In 1886 Joske was a resident medical officer at the Melbourne Hospital, and in the next year he occupied a similar post at the Alfred Hospital. During that year he distinguished himself by carrying out special work for typhoid fever patients, which was recognized by the presentation to him of a gold watch by the Central Board of Health. At a later date he wrote a thesis on typhoid fever, which gained for him the M.D. degree, which he took out in 1901.

On leaving the resident medical staff at the Alfred Hospital, Joske commenced private practice in Prahran. In that work he was closely associated with the Australian Natives' Association, and he was a very busy man for many years. He lived in the same house in Greville Street, Prahran, until in 1924 he removed to Huntingtower Road, Malvern, and concentrated attention on medical practice at 55, Collins Street, Melbourne. For a great number of years he was an active honorary physician at the Alfred Hospital, and at the time of his death was the senior consulting physician at that hospital. He was also the honorary medical officer to the Elizabeth Fry Retreat and the Freemasons' Homes, and from 1893 was an official visitor to the metropolitan hospitals. In 1897 he became a member of the Medical Board of Victoria and presided over the Medical Board from 1915 till his death. In addition he was for many years medical adviser to the Herald and Weekly Times, Limited, and the official medical attendant to the Victoria Amateur Turf Club.

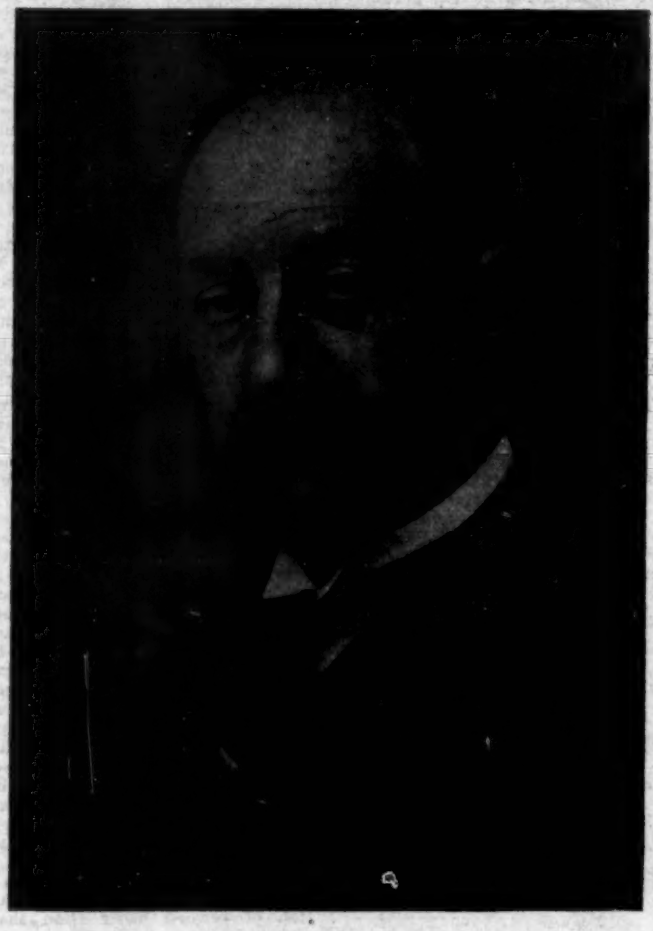
Dr. Joske was also a notable public figure. He was a distinguished patron of the humanities, a member of the Felton Bequest Trust and President of the National Gallery and Public Library. During the years at Prahran he became a justice of the peace and found the time to render service at the court house. His public activities were recognized officially when he became an Officer of the Most Excellent Order of the British Empire this year. He was an avid reader and kept himself well informed at all times. He read books extremely quickly and made himself conversant with their contents, retaining a vivid memory to the last. He was quick to recognize the outstanding merit of Rudyard Kipling and made a hobby of the collection of his works, leaving standing orders in England and in America to be supplied with each item as it appeared in the periodicals and in book form. In that way he accumulated an extremely interesting collection. He was one of the founders of the Melbourne Branch of the Kipling Society and occupied the position of president from its inception. Kipling's insistence on faithful attention to the job in hand without regard to its arduousness or the consequences and without expectation of reward other than that of a clear conscience struck a resonant note that appealed enormously to Joske.

This notice cannot be closed without reference to his home life. He was much devoted to his family, his garden and his dog. It was a happy home, and he was the central figure along with her who shared his married life for fifty years and is left behind to sorrow with their son, Dr. Esmond Joske, and four daughters and the younger members of the family. He lived a full life and his works live after him.

Dr. A. L. Kenny writes:

Alexander Sydney Joske was in the year ahead of me during our course in the medical school of the University of Melbourne. He was a good student, full of *joie de vivre*, and always ready for a quip or joke. With innate shrewdness he was a good judge of human nature and could comment very interestingly both upon his teachers and his patients. His career in the Alfred Hospital commenced in the bad Melbourne days of typhoid fever, the pre-

sewerage days, the days of the awful ironclads which disfigured St. Kilda Road returning to their northern suburbs in the early forenoon. Medical students nowadays rarely if ever see a typhoid fever case in the city hospitals. In those days these hospitals were overcrowded with such cases in the first quarter of the year. Because of the large open area surrounding it, the Alfred Hospital grounds were covered with marquees and tents filled with typhoid patients. Joske gained a large experience in the diagnosis, treatment and pathology of the disease, and utilized this in the preparation of the thesis which gained him his doctorate of medicine in the University of Melbourne. The work of Dr. Pettenkoffer, of Munich, in the ætiology and pathology of typhoid fever in that city, led him to organize a campaign to have the city sewered. The effect upon the incidence of typhoid fever was so immediate and striking that a similar campaign was inaugurated in Melbourne, and as a result the Government appointed a royal commission with Professor (afterwards Sir) H. B. Allen as chairman and George (afterwards Sir) Syme as Secretary. The work of this commission was so well done and so convincing that the sewerage of Melbourne was undertaken, and the effect was as immediate and striking as that in Munich.



Joske loved his many years of work as trustee and as chairman of trustees of the Public Library and National Gallery more than any of his other appointments. He missed no meetings and kept constant watch and ward over the institutions as if they were his own. A great lover of books and pictures, he was a cultured collector. No such collection of first editions of Rudyard Kipling's works as his exists in the southern hemisphere—I very much question whether its equal exists at Home. He knew and could quote his Kipling thoroughly. For very many years he was official visitor to the metropolitan mental hygienic institutions and member and chairman of the Medical Board of Victoria.

He was the best of good friends, as I can testify after fifty-seven years. His family life was most happy. As he would have wished, he died at work painlessly at the instant he was taking the chair at a meeting of the Medical Board of Victoria.

Sir James Barrett writes:

Dr. Alexander Sydney Joske represented one of the older group of practitioners who are disappearing. He was a personal friend of mine, but entered the medical school before I became a teacher in that department of the university.

He matriculated in 1880 and graduated in 1884 after the five years' course, which was then unknown in British universities. In 1901 he became a doctor of medicine.

He was appointed surgeon to the children's department of the Alfred Hospital in 1901, an office which he held until 1911, when he was appointed honorary consulting physician and held that office until his death. In addition, in 1902 he was appointed surgeon to out-patients as *locum tenens*, doing that work for approximately eighteen months.

As in the case of many medical practitioners who are really interested in social problems, he applied his technical knowledge and medical outlook to many fields in which such training rendered useful service.

He was appointed to the Medical Board of Victoria in 1897 and was elected chairman in 1915.

He was appointed a trustee of the Public Library, Museum and National Gallery of Victoria in 1903 and subsequently became treasurer (1933-1935), vice-president (1935-1937) and president (1937)—an office which he held until his death in 1939.

During the whole of this period he conducted a large practice and so demonstrated that professional work is not incompatible with most useful public service—a matter which Lord Horder has recently dealt with in an emphatic manner. Medical knowledge abuts on so many social problems that important work can be done, as in this case. In fact, if medical practitioners do not undertake it, the work cannot be properly done.

And so ends a useful life, the recollection of which may furnish some consolation to his family in their bereavement. Death is normal and natural, but none the less distressing to those who are left. *Requiescat.*

ARTHUR DAVID MAWSON.

We regret to announce the death of Dr. Arthur David Mawson, which occurred on October 23, 1939, at Seymour, Victoria.

NICHOLAS PHILLIPS ELLIOTT.

We regret to announce the death of Dr. Nicholas Phillips Elliott, which occurred on October 29, 1939, at Randwick, New South Wales.

Correspondence.

VENEREAL DISEASE AND WAR.

SIR: I was most interested in Sir James Barrett's letter published in a recent issue of your journal, regarding the incidence of venereal disease during the Great War. Since he wrote, hostilities between Britain and Germany have been reopened, and if we are to take Sir James's figures as an indication of venereal disease during the present crisis, it is apparent that there will be a high incidence again.

I would therefore like to carry Sir James's point a little further and indicate that an increase in venereal disease during the war must essentially precipitate an increase in *ophthalmia neonatorum*. I therefore make an appeal to all medical practitioners (and especially to obstetricians) in Australia to carry with them regularly a distinctive glass-stoppered bottle of fresh 1% silver nitrate drops and urge them to instil one drop into all babies' eyes at birth. Their positive cooperation in this way will materially help in a campaign which we hope will be launched soon throughout the whole of Australia for the prevention of blindness in every age of life.

The report of the Rotunda Hospital, Dublin, ending in October, 1938, contains a comment by Mr. L. B. Sommerville-Large, the hospital ophthalmologist. During the previous three-year period there had been 8,136 births in this hospital and no case of *ophthalmia neonatorum*, and 4,983 births in the district with only four cases of *ophthalmia neonatorum*. Perhaps some practitioners are not very conversant with the Rotunda method of prophylaxis, so I will detail here the routine of that hospital, which was given to me two years ago by Mr. Sommerville-Large, when I was in Dublin, and which I have had carried out ever since in Hobart. It is as follows:

Routine treatment of both eyes of all babies at birth.

1. Immediately the head is delivered the outside of the lids should be wiped with a dry sterile swab, using a separate swab for each eye.
2. When delivery is completed the baby should be confined by a towel wrapped round the body to prevent infection being carried to the eyes by the baby's fingers.
3. Immediately after birth one drop of silver nitrate, 1% (not 2%), should be instilled into each eye with a dropper. If the mother is known to have a vaginal infection a further drop is instilled into both eyes following bathing.
4. Should either eye subsequently discharge, the baby should be seen at once by an oculist.
5. The above routine requires the services of two experienced persons, one to hold the head, the other to instil the drops.
6. The solution of silver nitrate 1% should be kept in a small "Remco" drop bottle of such a style as to be recognized by touch as well as by sight.
7. These silver nitrate 1% drops should be renewed at least once a month, as they are liable to decompose and cause reaction in the conjunctiva.

I hope these simple instructions will be helpful to the medical profession of Australia, and if they are carried out assiduously they will give figures which will compare more than favourably with the magnificent result at the Rotunda Hospital, Dublin.

Yours, etc.,
J. BRUCE HAMILTON.

174, Macquarie Street,
Hobart,
October 21, 1939.

THE FUTURE OF MEDICAL PRACTICE.

SIR: The urgent need for efficiency in a time of national emergency brings to light many weaknesses in our social organizations, which have previously been passed tolerantly over; and in the ensuing reorganization much that is purely traditional is swept away if it cannot fit in with the scheme of efficiency, however much it may have been treasured in the past. Moreover, though the emergency passes, the impetus to planning does not, but remains to constitute a permanent step forward in the social scheme.

No side of the community life is exempt from this process; and in our own sphere the generation who were young men in 1914 have seen the hospital system drastically changed. Hospitals which were in our young days local charities, locally financed and locally governed, are now recognized to be part of a planned national service. They are increasingly financed by a central body, and increasingly accept, and look for, supervision in management from the same central authority. He would be a very bigoted conservative indeed who would now regret that change.

The new generation after this war will see the same process taking place with regard to the medical profession. For many years now, men in our own profession, and even more perhaps outside it, in political circles, have been drawing attention to the weaknesses of the present medical system; and threats of political action to alter it have been made more than once. Under the stress of this new war emergency those weaknesses have become and will become still more apparent, and it appears to me to be unquestionable that our traditional individualism will no longer be allowed to stand in the way of the needs of a more truly national service. Personally I feel glad that the greater part of my own professional life has been passed in the days of "private practice". But I am pretty sure that my son will pass his in quite other circumstances.

This letter is not a call for action in the matter, but a call for thought, so that when action does arise it may be wise. Its purpose is to urge those men among us who think beyond the moment; those men who have sons following or proposing to follow in their footsteps; who are interested in the future of medicine and the future careers of the young men now at the universities who are starting their medical lives; to urge them to give deep thought to this question of the nationalization of the profession. Not in the sense of organizing resistance against it, nor of evolving expedients whereby they may themselves personally escape from its effects; but to see if we within our own ranks cannot devise some plan to meet future social needs, whereby the transition in status of our profession from that of private traders to that of a national service can be made with a minimum loss of the professional spirit—the keenness for knowledge, the willingness to work for work's sake, the pride in work well done—which has enabled us to maintain in a commercialized world so high a standard that our faulty organization has been allowed to continue long after other less important services have been "rationalized".

Yours, etc.,
ARTHUR E. BROWN.

Colac,
Victoria,
October 24, 1939.

Analytical Department.

"POLYHÆMEN."

"POLYHÆMEN" is the name given by G.P. Pty. Ltd. to a tablet containing exsiccated ferrous sulphate and vitamin B_1. The manufacturers state that each tablet contains:

Iron (ferrous)	1·5 grains.
(*Ferri Sulphatis Exsiccati*, 4·5 to 5·5 grains.)	
Copper	0·01 grain.
Manganese	0·01 grain.
Cobalt	a trace.
Vitamin B_1	20 international units.

They hold that each "Polyhæmen" tablet is therapeutically equivalent to 30 grains of Blaud pill or to 50 to 60 grains of *Ferri et Ammonii Citras*.

Samples of "Polyhæmen" were submitted to our analyst, who reports that each tablet contains 1·5 grains of ferrous iron (as iron), 0·004 grain of copper (as copper) and 0·006 grain of manganese (as manganese). He concludes that 0·01 grain of anhydrous copper sulphate and 0·01 grain of manganous sulphate have been included in each tablet. The wording of the manufacturers' formula suggests that the copper and manganese content is expressed as metal. The amount of manganese found is somewhat greater than would be found if the only manganese found was that added as manganous sulphate. Manganese, however, is a common impurity in ferrous sulphate, and no doubt some of the manganese found comes from this source. Our analyst was unable to detect cobalt in the tablets; but this is not surprising, since there is no way of detecting cobalt in such small amounts (associated with 40,000 times its own weight of iron).

If it is understood that in the manufacturers' formula copper reads as copper sulphate and manganese as manganous sulphate, it may be accepted as correct.

"Polyhæmen" may therefore be accepted as providing ferrous iron and vitamin B_1 in a form suitable for use in the treatment of anæmias.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Basil-Jones, Brian James, M.B., B.S., 1939 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Diary for the Month.

Nov. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 10.—Queensland Branch, B.M.A.: Council.
Nov. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 14.—Tasmanian Branch, B.M.A.: Branch.
Nov. 15.—Western Australian Branch, B.M.A.: Branch.
Nov. 21.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 22.—Victorian Branch, B.M.A.: Council.
Nov. 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 24.—Queensland Branch, B.M.A.: Council.
Nov. 24.—Tasmanian Branch, B.M.A.: Council.
Nov. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 30.—South Australian Branch, B.M.A.: Branch.
Nov. 30.—New South Wales Branch, B.M.A.: Branch.
Dec. 5.—New South Wales Branch, B.M.A.: Executive and Finance Committee, Organization and Science Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii-xx.

CHILDREN'S HOSPITAL (INC.), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
DEPARTMENT OF PUBLIC HEALTH, PERTH, WESTERN AUSTRALIA: Medical Officer.
DEPARTMENT OF PUBLIC SERVICE, PERTH, WESTERN AUSTRALIA: Junior Medical Officer.
ROYAL HOBART HOSPITAL, TASMANIA: Resident Medical Officer.
ROYAL MELBOURNE HOSPITAL, VICTORIA: Honorary Officers.
SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.
WOMEN'S HOSPITAL, MELBOURNE, VICTORIA: Research Scholar.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
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